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LECTURES  
ON  
PRACTICAL MEDICINE AND PATHOLOGY

BY  
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## INFLAMMATION.

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THE phenomena which are embraced under the name of inflammation are :

Changes in the circulation of the blood.

Escape of the elements of the blood from the vessels.

Degeneration or death of tissue.

Growth of new tissue.

The growth of pathogenic micro-organisms and the formation by them of toxic substances.

These different morbid changes occur either separately or combined in a variety of ways.

We have, therefore, to consider, first, separately degeneration, necrosis, transudation, congestion, emigration, production, and the growth of micro-organisms ; and, secondly, the different combinations of these morbid processes.

### DEGENERATION.

*Definition.*—We mean by this term changes in the substances which compose the cells of the viscera, the nerve-fibres, and the muscular fibres. We do not include the so-called waxy, hyaline, fatty, glycogenic, mucous, colloid, and calcareous degenerations.

*Etiology.*—It is characteristic of such a degeneration as this that it does not occur by itself, but is always caused by the presence of some toxic substance. The poison may be an inorganic one, such as arsenic, phosphorus, or mercury ; or an organic one, such as is produced by the growth of pathogenic micro-organisms. How the poisons are carried to the different parts of the body, and in what way the changes in the cells are effected, we do not know.

The poisons of the different infectious diseases vary as to the part of the body which they habitually select. In typhoid and typhus fevers the muscles are degenerated ; in diphtheria the

Active congestion is often followed by exudative inflammation. Chronic congestion is often followed by productive inflammation.

#### EMIGRATION.

The escape of the white blood-cells from the capillaries and veins is usually associated with the transudation of blood-serum, but may occur without it.

One cause for emigration is the presence in the tissues near the blood-vessels of substances produced by bacteria which are positively chemotactic, that is, of substances which attract white blood-cells toward them.

It is also found that a variety of irritating substances in the tissues are capable of causing an emigration of white cells. Apparently whenever the emigration of white blood-cells is very large it is due to the presence of pathogenic bacteria, especially the streptococci and staphylococci.

The white blood-cells which have emigrated into the tissues may remain for a time as pus-cells and afterward degenerate and be absorbed. Or they may change into connective-tissue cells and form permanent new tissue. Furthermore these cells are capable of taking into themselves bacteria and other foreign bodies, and it is probable that in this way they may be of use in limiting infection.

It is also to be noticed that when a local emigration of white blood-cells is caused by bacteria there is at the same time an increase in the number of white cells in the blood throughout the body—leucocytosis.

#### PRODUCTION.

With or without other inflammatory changes there may be a growth of new tissue. This tissue follows the general type of connective-tissue cells and basement substance. It is known by the names of granulation tissue, round-celled tissue, connective tissue, fibrous tissue, tubercle tissue, etc. As a rule the more acute the process the greater the number of cells; the more chronic the process the greater the quantity of basement substance.

In an acute inflammation the production of new tissue may occur by itself, but is more frequently associated with exudation and emigration. In chronic inflammations the growth of new

tissues is often not attended with congestion, emigration, or exudation.

Whenever in any inflammation there is at the first a production of new tissue that inflammation regularly goes on to assume the sub-acute or chronic form.

#### MICRO-ORGANISMS.

Concerning the whole subject of micro-organisms the student should read the article on "The Biology of Bacteria, Infection, and Immunity," by Dr. Welch in the "American Text-book of the Theory and Practice of Medicine."

As regards the relationship of micro-organisms with inflammation, it has been demonstrated that the organisms themselves and the products of their growth are capable of causing exudation, emigration, degeneration, necrosis, and the growth of new tissue. They seem to be of most importance in connection with the purulent and necrotic inflammations.

The particular form of inflammation excited by the organisms seems to depend upon the number of organisms present and the virulence of the toxins which they produce.

In the severer local inflammations, which are accompanied by fever and other constitutional symptoms, these symptoms are principally due to the toxins produced by the growth of the organisms.

In classifying and naming the different forms of inflammation it is convenient to name them according to the most prominent of the different changes which go to make up the whole process.

We distinguish :

1. Exudative inflammation.

- (a) Simple exudative inflammation.

- (b) Purulent exudative inflammation.

2. Productive inflammation.

- (a) Simple acute productive inflammation.

- (b) Productive inflammation with exudation.

- (c) Chronic productive inflammation.

3. Necrotic inflammation.

1. *Exudative Inflammation*.—An exudative inflammation is one characterized by the presence of an exudate—serum, fibrin, and pus. The production of such an exudation may or may not be attended with changes in the inflamed tissues. The process may

run an acute, a subacute, or a chronic course. The character of the inflamed tissue, whether connective tissue, a mucous membrane, or a viscus, modifies the character of the inflammation.

We distinguish, therefore :

(a) Simple exudative inflammation.

(b) Exudative inflammation with large quantities of pus.

(a) So far as the exudate is concerned, we know by direct observation what it is and how it finds its way into the tissues.

In the inflamed tissue there is first a dilatation of the arteries, veins, and capillaries, and an increased rapidity of the circulation of the blood. Then the blood-current becomes slower ; while white blood-cells accumulate in the veins and capillaries, and adhere to their walls. This is the condition of "acute congestion," and the morbid process may not advance beyond this. If it does advance farther, the white blood-cells change their shape, find their way between the endothelial cells of the blood-vessels, through their walls, and appear on the outside of the vessels in the tissue. This is called "emigration." Red blood-cells in smaller numbers may also pass through the walls of the capillaries and veins, and this is called "diapedesis." At the same time the plasma of the blood transudes through the walls of the vessels and infiltrates the tissues as serum ; while by the union of substances contained in the blood-plasma and in the white blood-cells, fibrin is formed.

In this manner are elaborated the inflammatory products—pus, serum, and fibrin. The pus-cells are emigrated white blood-cells ; the serum is part of the plasma of the blood ; the fibrin is produced by a union of the fibrinogen in solution in the blood-plasma with substances contained in the white blood-cells, and appears coagulated in the form of granules, amorphous masses, or a reticulum.

The relative quantity of pus, serum, or fibrin varies in different cases.

In "simple exudative inflammation" we find no other morbid changes than the congestion and the exudation ; the tissues remain unaltered. When the inflammation has subsided all the parts return to their natural condition.

It is in connective tissue that a simple exudative inflammation is seen in its most typical form. The structure of connective tissue is simple—a basement substance, cells, blood-vessels, lym-



phatics, and nerves. The inflammation is attended with an increased quantity of blood in the vessels, more or less swelling of the basement substance and cells, and the exudation collected in the natural cavities of the tissue.

The structure of the mucous membranes is more complex. They are all composed of a layer of epithelium, of a connective-tissue stroma containing the blood-vessels, nerves, and lymphatics, and of glands which produce mucus. The inflammation not only causes the same congestion and exudation in the stroma, but there are also changes in the epithelium and the glands. In the epithelium there is a more active desquamation of old cells and growth of new cells; sometimes superficial ulcers are formed. The function of the mucous glands is interfered with. At first the production of mucus is stopped, later it is increased and altered. The increased production of mucus is regularly attended with a diminution of the congestion and swelling of the mucous membrane. Such an inflammation in a mucous membrane is often called "acute catarrhal inflammation."

The viscera are composed of a connective-tissue stroma containing the blood-vessels, lymphatics, and nerves, and of cells. The cells are peculiar to each viscus, and are concerned in performing the functions of the viscus.

The principal changes effected by the inflammation are the congestion and consequent swelling and the inability of the visceral cells to perform their proper functions. The quantity of exudation is small.

(b) In exudative inflammation with an excessive quantity of pus, or purulent inflammation, the excessive number of pus-cells may, or may not, be accompanied by serum and fibrin. Such an excessive number of pus-cells is due to an increased emigration of white blood-cells.

The inflammation is of a more severe type than a simple exudative inflammation. The pus-cells infiltrate connective tissue, they are mixed with the serum in the serous cavities and with the mucus on the surfaces of the mucous membranes, but they do not form abscesses.

## II. *Productive Inflammation:*

(a) Simple acute productive inflammation.—In this form there is no exudation, no serum, fibrin, or pus. Congestion is sometimes visible after death, but by no means always. The inflammatory product consists of new cells formed from the old

connective-tissue cells. The pia mater and the peritoneum offer the best examples of this form of inflammation.

(b) Productive inflammation with exudation.—In this form of inflammation the changes in the blood-vessels, the exudation and emigration, the formation of serum, fibrin, and pus are well marked, but in addition there is from the first a growth of new tissue. This new tissue at first consists principally of cells, later a basement substance and blood-vessels are added. This form of inflammation has a marked disposition to continue for a long time in a sub-acute or chronic form.

In connective tissue the serum, fibrin, and pus are found in varying quantities. The new tissue forms thickenings, and adhesions.

In the mucous membranes the inflammation involves the stroma, and it is in the stroma that the exudation is infiltrated and the new tissue formed. The glandular coat may remain unchanged, or be the seat of catarrhal inflammation.

In the viscera the quantity of the inflammatory product varies. The new tissue is formed in the stroma. The visceral cells undergo more or less atrophy or degeneration.

(c) In chronic productive inflammation the inflammatory product is round-celled tissue, granulation-tissue, or connective tissue. In some cases this is the only change, in others there is added an exudation from the blood-vessels, or degeneration of cells. The new tissue that is formed may degenerate or become calcified.

*In connective tissue* this form of inflammation produces thickenings and adhesions, and serum in the serous cavities.

*In mucous membranes* the growth of new tissue is in the stroma. This is thickened, either diffusely, or in the form of polypoid growths. The layer of epithelium may be thickened or thinned. The mucous glands are atrophied, or hypertrophied, or become cystic. The production of mucus is diminished, or increased, or altered. This condition in the mucous membranes is commonly called a "chronic catarrhal" inflammation.

*In the viscera* there is a growth of indifferent tissue, or of connective tissue, in the stroma. The visceral cells are compressed, or degenerated, or fatty, or disappear. The functions of the viscus are seriously interfered with. In the viscera this is often called an "interstitial" inflammation.

The most marked features of this form of inflammation are its

slow course and its tendency to continue. The lesions of chronic productive inflammation, especially in old persons, are by some believed to be due to chronic degeneration.

III. *Necrotic Inflammation*.—In this form of inflammation, in addition to the congestion and exudation, there is added death or degeneration of parts of the tissues in which the inflammation exists. This character of the inflammation is given to it by the presence and growth of pathogenic bacteria. The bacteria regularly present are the staphylococcus pyogenes aureus and albus, and the streptococcus pyogenes. They frequently occur together in the same inflammatory process.

Such an inflammation, when it occurs in connective tissue, produces abscesses. A circumscribed portion of tissue is congested, infiltrated with serum, fibrin, and pus, and parts of the tissue die. The dead tissue softens, breaks down, and cavities are formed which contain serum, pus-cells, and portions of dead tissue.

In mucous membranes there are congestion, exudation of serum rich in fibrino-plastic substances, emigration of white blood-cells, and necrosis of tissue.

The fibrin infiltrates the stroma, and coagulates on the surfaces of the mucous membranes so as to form false membranes. The pus-cells are entangled in the fibrin. The necrosis involves only the epithelium, which passes into the condition of coagulation necrosis, and forms part of the false membranes; or it involves also the stroma. The death of the epithelium forms superficial erosions, that of the stroma ulcers of varying size and depth.

Such an inflammation of the mucous membranes is called "croupous" or "diphtheritic." We also find with catarrhal and productive inflammations circumscribed necrosis of the epithelium.

In the viscera we find congestion, exudation of albuminous serum, and emigration of white blood-cells. In addition there may be degeneration or death of the visceral cells; or death of portions of the stroma with groups of cells, and the formation of abscesses.

This variety of inflammation is of severe type, is accompanied with marked symptoms, and after it has subsided, leaves changes in the tissues.

In connective tissue the cavities of the abscesses are filled

first with granulation tissue, and afterward with cicatricial tissue.

In the mucous membranes the dead epithelial-cells may be replaced by new cells of the same kind, but the ulcers formed by the death of the stroma have to be filled first with granulation tissue, and later by cicatricial tissue.

In the viscera the degenerated cells may be replaced by new visceral cells, but the abscess cavities are filled first with granulation tissue, and later with cicatricial tissue.

The treatment of the different forms of inflammation is a matter of practical importance.

In exudative inflammation the most efficient treatment is the use of local means which cause contraction of the blood-vessels. Such a contraction can be caused by the application of cold, of heat, and of astringents. Somewhat less certain as a local treatment, although apparently often of service, is the use of counter-irritation, or of local blood letting, by which the congestion of the inflamed tissues seems to be relieved. Whether a local inflammation is favorably affected by general blood-letting is uncertain.

In some cases, by no means in all, this form of exudative inflammation seems to be favorably affected by the use of drugs. If we dilate the small arteries throughout the body, it seems probable that we can diminish the congestion of any one circumscribed part of the body, and so we give aconite, veratrum viride, and nitro-glycerine.

In the mucous glands we see that, as a larger quantity of mucus is produced the congestion subsides, and so we give drugs which are likely to increase the production of mucus, such as ipecac, antimony, and pilocarpine.

Then there are certain drugs which we use empirically : calomel or sulphate of magnesia in small doses frequently repeated for a few hours ; opium given for a longer time ; in the case of some of the mucous membranes, large doses of ipecac.

In the forms of exudative inflammation with death of tissue, suppuration, and the growth of pathogenic bacteria, it has been conclusively shown that, if the bacteria can be excluded the inflammation will not assume this character. When, however, such an inflammation is once established, it is not easy to destroy the bacteria and subdue the inflammation by the local use of germicides. On the other hand, the evacuation of collections of

pus and of serum with antiseptic precautions and complete closure of the wound, is regularly followed by subsidence of the inflammation.

When a production of new tissue is added to an exudative inflammation, we employ the same means of treatment as for an exudative inflammation, but without the same probability of success. Such inflammations are much more likely to become chronic, or to cause permanent changes.

The chronic productive inflammations are much less amenable to treatment. The internal administration of drugs, except in the case of syphilis, is of little avail. We rely principally on local treatment, regulation of the diet and mode of life, and climate; but it has long been believed that preparations of mercury, and the iodide of potash, are of use in some examples of this form of inflammation.

#### THE PIA MATER.

The external surface of the brain is overlaid by a connective-tissue membrane which covers the convolutions, dips down into the sulci, and extends into the ventricles. This membrane is abundantly supplied with blood-vessels, and from it numerous vessels extend into the brain, so that any disturbance in the circulation of the blood in the pia mater involves a disturbance in the circulation of the blood in the brain also.

The connective tissue which makes up the pia mater is arranged in a series of membranes and fibres reinforced by elastic tissue, so arranged as to form a spongy membrane containing numerous cavities more or less filled with fluid. These cavities are continuous with the perivascular spaces which surround the vessels that pass from the pia mater into the brain. The outer layers of the pia mater are the most compact, and are covered on their outer surface by a continuous layer of endothelial cells. This external layer of the pia mater is often described as a separate membrane called the "arachnoid," but it is really only part of the pia.

The deeper layers of the pia contain the blood-vessels. The membranes and fibres which compose the pia mater are partly coated with cells, which have irregular and delicate cell-bodies and large, distinct nuclei.

In all inflammations of the pia mater the inflammatory prod-

ucts regularly collect in the spaces within it. Only occasionally do we find them on its free surface.

The pia mater is frequently thickened, opaque, and white, either in diffuse patches, or along the course of the vessels. In other cases, single or multiple white spots of the size of a pin's head, or smaller, may be seen in the membrane, not appreciably elevated above the surface, but due to localized thickening. These slight opacities of the pia mater are commonly believed to be dependent upon repeated congestions of the membrane, or upon chronic meningitis, but there is no evidence that this is always the case. They are most frequently found in old persons, but may exist at any age, and do not necessarily indicate the pre-existence of disease, although similar appearances are common in the chronic insane and in drunkards.

CONGESTION AND ANÆMIA.—There can be little question that the quantity of blood contained in the vessels of the pia mater varies at different times, and that any considerable increase or diminution of the quantity of blood is likely to give cerebral symptoms. In the human subject, however, our knowledge of this subject is as yet indefinite.

ŒDEMA.—The quantity of serum beneath the pia mater and in its cavities is very variable. It may accumulate as a result of atrophy of the brain-substance or of venous hyperæmia, and may be accompanied by œdema of the brain-substance also. It is not infrequent to find in hospital patients suffering from chronic nephritis, cardiac or pulmonary disease, or chronic alcoholism, a very considerable quantity of serum in this situation, and yet the patient has been free from cerebral symptoms. In other cases, again, this same effusion affords the only explanation of grave cerebral symptoms.

#### ACUTE MENINGITIS.

*Lesions.*—The ordinary form of acute inflammation of the pia mater is the exudative, but occasionally the inflammation is of pure productive type without exudation. Either form of inflammation, however, is attended with the same symptoms.

In acute productive, or cellular, meningitis the pia mater is congested, its surface is dry and lustreless, it is somewhat opaque, it is not at all infiltrated with serum. These changes in the gross appearance of the membrane are not marked and are easily overlooked, but the minute changes are more decided. There is an

abundant production of cells somewhat resembling the cells which coat the surfaces of the membranes and fibres which make up the pia mater. This cell-growth is general, involving the pia mater over most of the surface of the brain, but the cells are much more numerous in some places than in others. The inflammation, then, is one which results in the production, not of fibrin, serum, or pus, but of new connective-tissue cells.

In acute exudative meningitis there is an accumulation of serum, fibrin, and pus in the meshes of the pia mater and along the course of the vessels, rarely on the surface of the pia. Sometimes one, sometimes another of these exudations preponderates, giving rise to serous, fibrinous, or purulent forms of the inflammation. The absolute quantity of the exudation varies greatly. The quantity of exudation may be so small that the pia looks nearly normal to the naked eye, and the pus and fibrin can only be seen with the microscope. More frequently the quantity of exudation is considerable, and often very large, even sufficient to flatten the convolutions of the brain. The cortical portion of the brain may be œdematous, or degenerated, or infiltrated with minute hemorrhages. Very often the inflammation extends to the ventricles, which then contain purulent serum. In children the ventricular lesion is regularly well marked, the ventricles are dilated, and contain large quantities of purulent serum. In adults such a distention of the ventricles occurs less frequently. The inflammation of the ventricles may persist for days and weeks after the subsidence of the meningitis.

The exudation may cover the whole surface of the brain, or be confined to the base, or to the convexity. It often extends down in the pia mater of the cord, and the roots of the cranial nerves may also be involved.

In the purulent forms of acute meningitis, bacteria have been found. The ordinary streptococcus of purulent inflammation, the diplococcus pneumoniae, the diplococcus intra-cellularis, a bacillus like that of typhoid fever, have been described. In an epidemic of cerebro-spinal meningitis Bonome has found a special form of streptococcus.

*Causes.*—Acute meningitis occurs under four entirely different conditions. It is produced by injuries, by the extension of inflammations from the cranial bones, the ear, the dura mater, and by infection from streptococcus inflammation in other parts of the body.

It complicates pneumonia, rheumatism, nephritis, and many of the infectious diseases.

It occurs as a primary lesion without discoverable cause.

It is the characteristic lesion of the infectious disease called epidemic cerebro-spinal meningitis.

*Symptoms.*—THE IDIOPATHIC CASES. It is difficult to distinguish these cases from sporadic cases of cerebro-spinal meningitis, and our descriptions of idiopathic meningitis are probably very much modified by our observations of the epidemic disease. The idiopathic form seems to be more common in children.

The invasion of the disease may be preceded by a prodromic period characterized by conjunctivitis, nausea and vomiting, headache, irritability of temper, sleeplessness, and general malaise; or it may be sudden, with fever, headache, convulsions, delirium, and vomiting. The cases vary as to which one of these symptoms is the prominent one at the outset. Either the headache, the convulsions, the delirium, or the vomiting may be especially marked.

When the disease is fairly developed the headache is continued and severe. Throughout the disease perhaps the most prominent symptoms are restlessness and stupor. The restlessness ranges from irritability and sleeplessness up to violent delirium; the stupor from apathy up to coma. In many patients the restless condition occupies the earlier days of the disease, and the stupor is gradually developed later. In others the restlessness and stupor alternate; or either one may predominate throughout the disease.

Involuntary contractions of groups of muscles, especially of those of the face, are often present. Unilateral or general convulsions occur in some of the cases.

Localized or general hyperæsthesia of the skin may exist. If the inflammation extends down to the pia mater of the cord, there is tenderness and contraction of the muscles of the neck.

As the inflammation involves the roots of the cranial nerves, photophobia, blindness, strabismus, painful hearing, and deafness are developed.

The vomiting which belongs to the outset of the disease may continue, or it may not begin until later.

The tongue is coated, and in bad cases becomes dry.

Constipation is the rule, but diarrhœa and involuntary movements may come on in the last days of fatal cases.



The urine is diminished in quantity ; it may contain a little albumin and a few casts.

The temperature usually runs between 100° F. and 104° F. ; it is apt to follow an irregular course.

The pulse is at first rapid, then slow, and, in the fatal cases, again rapid and weak, but it may be rapid throughout the disease.

In children the course of the disease may be the same as in adults. Often, however, the excessive development of the inflammation of the lateral ventricles and their distention with serum, causes a difference in the symptoms. In some children almost the only symptoms are a febrile movement and convulsions alternating with stupor. In others the course of the disease is like that of tubercular meningitis.

The duration of an acute meningitis is from thirty-six hours to four weeks. The ordinary duration is from seven to fourteen days.

The prognosis is bad, but not hopeless.

**SECONDARY ACUTE MENINGITIS.**—First there are the symptoms of the primary inflammation, and then those of the meningitis.

If the meningitis is secondary to an acute otitis there are first the fever, prostration, and severe pain belonging to the otitis. Then the patients become worse, the prostration is more marked, the temperature is higher, alternating delirium and stupor are developed, there are contractions of the muscles of the face, slow pulse, and finally coma. But it must be remembered that a severe acute otitis, especially in children, may give marked cerebral symptoms without meningitis.

If the meningitis follows a chronic otitis there will be the history of the ear trouble extending back for months or years ; then suddenly come on the symptoms of the meningitis—fever, headache, alternating delirium and stupor, coma.

**COMPLICATING MENINGITIS.**—The diseases which are liable to be complicated by meningitis are all of them capable of giving marked cerebral symptoms without any inflammation of the pia mater. When a complicating meningitis really is developed, the only difference is that the symptoms are more marked and approach more closely to those of an idiopathic meningitis.

*Treatment.*—The indications for treatment are to diminish the severity of an acute exudative inflammation, to alleviate the pain, and to nourish the patient.

The measures directed toward the inflammation are most efficacious during the early days of the disease. We employ continuous cold over the head by ice-bags or the rubber coil, sometimes blood-letting from the temples and the back of the neck, and the internal administration of calomel, sulphate of magnesia, opium, the iodide of potash, or ergot.

The pain and restlessness may be controlled by the bromides, chloral, or opium.

The patients are to be kept perfectly quiet in a darkened room, on a fluid diet, with the addition of alcoholic stimulants when the heart's action begins to fail. The bowels are kept open by mild laxatives or enemata.

#### CHRONIC MENINGITIS.

True chronic meningitis is a very real and serious lesion. It is not to be confounded with the simple opacities and thickenings of the pia mater which are so common in adult life.

*Lesions.*—Either the pia mater or the base of the brain alone may be inflamed (basilar meningitis), or the pia mater over the convexity alone or the entire pia mater, or circumscribed patches of the membrane. The pia mater is thickened and opaque, the thickening being sometimes very considerable. There is a formation of new connective tissue and a production of pus, fibrin, and serum; the relative quantity of these inflammatory products varies in different cases. Firm and extensive adhesions may be formed between the dura mater and the pia mater. Not infrequently the cortical portions of the brain participate in the morbid process, and we find an infiltration of small spheroidal cells around the blood-vessels, thickening of the walls of the vessels, and degenerative changes and atrophy of the brain tissue. New connective tissue may also form in the brain substance, and the latter may become adherent to the pia mater. The ventricles of the brain may be much dilated and distended with serum, their endyma may be thickened and roughened.

*Causes.*—Chronic meningitis may be caused by blows on the head, and by fractures and inflammations of the cranial bones. Sometimes the injury to the head antedates by several years the symptoms of the meningitis. It is often associated with pachymeningitis, with tumors of the brain, and with chronic endarteritis of the cerebral arteries. It is the direct result of syphilis.

It occurs with chronic nephritis, with chronic alcoholism, and in persons who are badly nourished and of dissipated habits. It is regularly found in the general paralysis of the insane.

*Symptoms.*—The course of the disease is that of an inflammation, chronic from the outset, and with exacerbations from time to time. The symptoms are at first obscure and intermittent, and vary much in different individuals as to their number and distinctiveness. So the diagnosis is always difficult and sometimes quite impossible.

The patients complain of more or less headache—continuous or intermittent. The mental faculties and the temper gradually deteriorate, and such a change may go on to complete insanity. From time to time there are attacks of unconsciousness, of stupor, of delirium, of muscular contractions or rigidity, of aphasia, of general convulsions, or of vomiting. After a time the nutrition is impaired, and there is a gradual loss of flesh and strength. At the times when the symptoms are most marked there may be a moderate rise of temperature.

Chronic meningitis usually lasts for a number of years, but it may happen that the early symptoms are slight and that at some time severe symptoms are suddenly developed, as if of an acute cerebral lesion.

The patients become insane, or die with marked cerebral symptoms, or in a condition of emaciation and exhaustion.

*Treatment.*—The first point to determine is whether the patient has had syphilis. If this is the case, the use of mercury and the iodide of potash may be of much service. In the cases due to other causes there seems to be no direct treatment for the meningitis. The improvement of the general health, and the alleviation of symptoms must be attempted, but as a rule the patients get worse.

#### TUBERCULAR MENINGITIS.

Tubercular inflammation of the pia mater behaves differently in children and in adults, so that it is necessary to describe the disease separately according to the age of the patient.

I. TUBERCULAR MENINGITIS IN CHILDREN. *Lesions.*—The dura mater is usually unchanged, but there may be miliary tubercles on its inner surface. The surface of the brain is flattened by the pressure of the fluid which distends the ventricles. The inflammatory process is a combination of tubercular and of exudative

or cellular inflammation, either one of which may predominate. The tubercles are very small and transparent, or large and white. They are composed of simple aggregations of small cells, or of well-formed tubercle tissue arranged around blood-vessels. The vessels show the changes of obliterating endarteritis. The cellular inflammation produces new connective-tissue cells, the exudative inflammation produces serum, fibrin, and pus. The inflammation is often confined to the base of the brain, but may extend over its entire surface, or be confined to the convexity. The tubercles are most numerous in the pia mater over and in the sulci.

The ventricles of the brain are much dilated and distended with serum. Their ependyma is thickened and studded with very small miliary tubercles. The brain tissue around the ventricles is softened. There may be similar lesions in the pia mater of the cord.

Such a tubercular meningitis is regularly, but not always, only part of an acute general tuberculosis with similar lesions in many different parts of the body.

*Causes.*—The children affected are usually under five years of age. There is often a family history of tubercular disease. The meningitis is regularly only one of the lesions belonging to an acute general tuberculosis. Such a general tuberculosis may be a primary disease, or secondary to a localized tuberculosis.

*Symptoms.*—It is important to remember that the tubercular meningitis is usually only part of a general tuberculosis, and that the intensity of the inflammation of the pia mater varies much in different cases. So we find that in some cases the symptoms are more like those of a general tuberculosis, in others like those of a meningitis, and that the meningitis may have the characters of an acute or a sub-acute inflammation.

There may be a prodromic period, probably due to the development of the general tuberculosis, which usually lasts only for a few days, but may be protracted for several weeks. During this period there are loss of flesh and strength, slight evening fever, irritability, sleeplessness, frontal headache, vomiting, constipation, and a coated tongue. These symptoms are not continuous, but are interrupted by periods of improvement.

When the disease is established the child lies in bed, the face flushed, in a condition of alternating stupor and delirium, either one of which may predominate. During the periods of restless-

ness the child seems to have severe pain in the head ; there is photophobia and hyperæsthesia of the skin. There is often frequent and active vomiting. The child rolls its head, moans and cries out, it may be actively delirious, there may be one or more general convulsions. During the period of stupor it lies quietly in bed, taking no notice, but apparently placid and comfortable. The bowels are constipated. The pulse is slow, in proportion to the temperature, but irregular. The breathing also is irregular. There is a febrile movement which runs a very irregular course, but is not necessarily at any time very high. The urine is scanty and contains albumin.

At the end of the first week, or a little earlier or later, strabismus, inequality of the pupils, and ptosis are developed.

In the second week the stupor deepens into coma, which may be continuous or alternate with active delirium. The head is drawn back, there are convulsive movements, or paralysis, or rigidity of the limbs and face, which may be transient or permanent. There may be automatic movements of the arms and legs. The patients constantly pick at the nose and lips. The pupils are dilated, and vision is lost. The pulse becomes very rapid and feeble. The course of the disease is interrupted by short periods of improvement ; it lasts for from two to four weeks. The patients die in an attack of convulsions, or they become very feeble and emaciated ; the urine is suppressed, the pulse and breathing are very rapid, the temperature remains high, or falls below the normal. This is the history of the cases with general tuberculosis and a moderate meningitis.

With a more intense meningitis the cerebral symptoms are more active, the temperature is higher, and the disease runs its course within a week.

If the meningitis is confined to the convexity of the brain there is no strabismus and little vomiting. The headache, delirium, convulsions, and rigidity of the muscles are the chief symptoms. The disease runs its course within a week.

*Treatment.*—The treatment varies with the acuteness of the meningitis. In cases which run an active course, like that of an acute meningitis, we employ the same methods of treatment as for that disease. In the cases which behave more like general tuberculosis with subacute cerebral symptoms, the nursing, feeding, and alleviation of symptoms constitute the entire treatment.

As a rule, the patients do not recover, but from time to time

we see cases which behave like tubercular meningitis and yet do recover. Whether in these cases the meningitis really is tubercular it is impossible to say.

2. TUBERCULAR MENINGITIS IN ADULTS.—The changes in the pia—the combinations of tubercular, exudative, and cellular inflammation—are the same as in children. There is, however, one important difference between adults and children. In children the inflammation of the ependyma of the ventricles, the distention of the ventricles with serum, and the consequent compression of the brain are the rule ; while in adults they are the exception.

In adults, as in children, the tubercular meningitis may only be part of a general tuberculosis. But more frequently it occurs as a localized tubercular inflammation, either primary or secondary to a localized tubercular inflammation of some other part of the body.

*Causes.*—The greatest liability to the disease seems to be between the ages of fifteen and twenty-five years. Persons who inherit the tubercular disposition, or who have chronic pulmonary phthisis, are especially liable to the disease. It may very well happen that the pulmonary lesion is small and insignificant.

*Symptoms.*—It is convenient, in describing the symptoms of tubercular meningitis in adults, to divide the cases into three clinical groups :

(1) Cases of acute general tuberculosis with tubercular meningitis, but without the symptoms of meningitis. The history of these patients belongs to general tuberculosis.

(2) Cases with well-marked symptoms of meningitis, which come on more or less suddenly, and are developed in persons whose health was apparently good up to the time of the attack. Of these cases there are a number in which a localized tuberculosis has existed in some part of the body, but has given so little trouble that it has not been recognized, so that when the secondary meningitis is developed it seems to be a primary inflammation. In other cases, however, the meningitis is a primary, localized, tubercular inflammation.

In all these cases the invasion of the symptoms may be sudden or gradual. If it is sudden there are chill, fever, headache, vomiting, and so much prostration that the patient is at once confined to bed. If it is gradual the patients do not feel sick enough to go to bed for several days. They have a little fever, headache, loss of appetite, and general malaise.

When the disease is fairly established the headache is severe and continuous. The patients are sleepless, restless, and go on to mild or active delirium. The delirium alternates with stupor, or coma. There are conjunctivitis, photophobia, ptosis, strabismus, involuntary contractions of the muscles of the face, arms, and legs. From two to five days before death there may be permanent hemiplegia, monoplegia, or facial paralysis. Hyperæsthesia of the skin is present in some cases. Vomiting is often persistent and troublesome. A few days before death there may be difficulty in swallowing. In some cases there is pain and contraction in the muscles. The urine contains albumin and casts. The temperature may run low or high, and follows a very irregular course. Generally speaking, the cases of short duration have high temperatures, and those of long duration low temperatures. The pulse is irregular, sometimes slow, sometimes rapid.

The symptoms may be intense, continuous, and the patients die in from seven to fourteen days; or they may be less severe, with intervals of improvement, and the patients may go on for from thirty to fifty days.

(3) Cases with a well-marked history of pulmonary phthisis may at some time develop the symptoms of tubercular meningitis.

The prognosis of tubercular meningitis in the adult is regularly bad, but yet we see patients with characteristic symptoms of the disease who recover.

*Treatment.*—The indications for treatment are the same as in the tubercular meningitis of children.

#### INFLAMMATION OF THE CEREBRAL ARTERIES.

The arteries throughout the body are frequently the seat of chronic inflammation.

In the cerebral arteries the ordinary changes are as follows :

1. There is an increase in the size and number of the endothelial cells. This is best seen in the small arteries surrounded by miliary tubercles, or by small gummata.

2. There is a growth of new connective tissue, apparently originating in the endothelium, which narrows the lumen of the artery and may finally occlude it. The growth is composed of branching cells, small round cells, and basement substance ;

later the cells become smaller and less numerous, the basement substance denser.

The growth forms a ring on the inside of the intima which is not symmetrical, but is thicker in some places than in others. This is often called obliterating endarteritis.

3. There is a thickening of the inner coat beneath the endothelium. The change begins by a growth of cells and a splitting up of the basement substance in the intima immediately beneath the endothelium. Then there is a growth of basement substance with but few cells, which renders the inner coat thicker and thicker until the lumen of the artery is considerably narrowed. These changes may occur by themselves or there may be, in addition, thickening of the muscular and outer coats, replacement of the muscular coat by dense connective tissue, thinning of portions of the arterial wall and irregular dilatations, deposition of the salts of lime in the new tissue.

As a result of these changes the arteries are rendered very irregular channels for the passage of the blood—narrowed in some places, dilated in others, or completely occluded. The blood-supply of the corresponding portions of the brain is variable or cut off altogether.

The diseased arteries may rupture, with extravasations of blood. Portions of the brain may become softened from the occlusion or thrombosis of the arteries. Either small or large aneurisms may be formed.

The changes may be confined to the larger arteries at the base of the brain, or they may involve nearly all the branches of the cerebral arteries.

Marked cerebral symptoms are seen during life in persons in whom after death no changes are found in the brain, nothing can be made out except the endarteritis.

There may be similar changes in the arteries in other parts of the body, or the cerebral arteries are the only ones affected.

*Causes.*—Chronic endarteritis is especially common in persons over forty years old. It is frequently associated with chronic gout, constitutional syphilis, pulmonary emphysema, chronic Bright's disease, and chronic endocarditis. But it may also occur by itself and without discoverable cause.

*The symptoms* presented by these patients are at first in the form of temporary attacks, which last for hours or days, and then disappear. Such attacks are repeated at irregular intervals



during months or years, the patient presenting few or no cerebral symptoms during the intervals. As time goes on the attacks become more severe and more frequent. It is, however, possible for the patient to die during the first attack.

The temporary attacks are not all of the same character. There may be only headache, or anxiety, restlessness, and insomnia, or vertigo, or aphasia, or hemianopia, or loss of consciousness, or general convulsions, or spasmodic contractions of groups of muscles, or paralysis of one arm, of one leg, or of the whole of one side of the body, or loss of sensation in different areas of the skin.

The first attack may prove fatal, but the patient often continues to live for months or years. Even after recovering from the first attack, however, there is a perceptible change in the patient's mental and bodily condition, sometimes very marked. The attacks are repeated until finally the patient develops the symptoms of chronic meningitis, or becomes completely hemiplegic, or dies with general convulsions or coma.

*Treatment.*—Apart from antisyphilitic treatment in the proper cases, the most important points to enforce are abstinence from all alcoholic drinks, and exercise in the open air.

#### THE VENTRICLES OF THE BRAIN.

Inflammations of the pia mater are often accompanied by lesions of the ventricles of the brain. Less frequently we find lesions of the ventricles occurring by themselves, with little or no change in the pia mater. We distinguish :

Acute ependymitis.

Acute dilatation of the ventricles in adults.

The chronic hydrocephalus of children.

Chronic hydrocephalus due to injuries, or secondary to meningitis.

1. ACUTE EPENDYMITIS.—Of this we find two varieties: an acute and a subacute form.

In the acute form we find the ependyma congested, infiltrated with pus-cells, and coated with a layer of fibrin and pus. The ventricles contain purulent serum.

The patients exhibit a decided febrile movement, general convulsions, alternating stupor and delirium, local paralysis, and finally coma. The disease runs its course within a few days.

The symptoms resemble those of an acute tubercular meningitis.

So few of these cases have been observed that we are ignorant of the prognosis.

The treatment would seem to be the same as that for acute meningitis.

In the subacute form the ventricles are dilated and full of serum. The ependyma is thickened, the endothelial cells on its surface are multiplied, and there is a growth of cells around the blood-vessels.

The patients have headache; vomiting; at first photophobia, later dilated pupils; hyperæsthesia of the skin; an irregular rise of temperature, never very high, and sometimes below the normal; finally coma. The disease lasts for weeks or months. The symptoms resemble those of a subacute tubercular meningitis.

These cases again are so seldom recognized that we do not know how many of them may recover.

2. ACUTE DILATATION OF THE VENTRICLES IN ADULTS—SEROUS APOPLEXY.—*Lesions.*—Either one lateral ventricle, or both, or all the ventricles are dilated and filled with clear serum. The ependyma is white, somewhat thickened, its surface smooth or finely granular. The convolutions of the hemispheres are somewhat flattened.

*Causes.*—The disease is, in New York, not an uncommon one. It occurs both in strong and vigorous adults and in persons who have suffered from chronic diseases. In some persons there is a previous history of injuries to the head, or of mental or bodily overwork. In some cases the condition is complicated by chronic endarteritis, or chronic meningitis. It is said that there may be an obstruction of the veins or of the passages between the ventricles.

The disease may be confounded with cerebral apoplexy, embolism, chronic endarteritis, or tumors of the brain.

*Symptoms.*—The patients suffer from headache, noises in the ears, loss of memory, dulness of the mental faculties, slowness of speech, sleeplessness, disturbed digestion, sometimes temporary aphasia, or local paralysis. These symptoms may only last for a few hours, or they may continue for a number of weeks. At the end of this time some of the patients recover completely.

Others, however, either rapidly or gradually pass into the con-

dition of complete coma. In this condition they remain for a few days and then die, the temperature rising before death.

Others, on the contrary, develop an active delirium, blindness, internal strabismus, convulsive movements of groups of muscles, a febrile movement, and finally become comatose.

*Treatment.*—Recoveries from the disease are reported after treatment with mercury and potassium iodide.

3. THE CHRONIC HYDROCEPHALUS OF CHILDREN.—*Lesions.*—There is an accumulation of serum in the ventricles of the brain, so large in quantity that the brain is thinned, the dura mater stretched, and the cranial bones separated from each other. The ependyma appears to be normal, or is somewhat thickened.

*Causes.*—We are ignorant of the causes of this lesion. It seems to begin during intra-uterine life. It may reach a considerable development before birth, or remain latent for months or years after birth.

*Symptoms.*—If the disease is so much developed during intra-uterine life that the large head prevents the birth of the child, craniotomy must necessarily be performed.

If the disease is moderately developed during intra-uterine life, the child can be born alive. But it is at once evident that the head is too large; the child does not perform its functions well, and dies in a short time.

In many cases, at the time of birth the child shows no evidences of the disease, and it is not till after months or years that its symptoms are developed. Of these later developed cases we may distinguish a mild and a severe form of the disease.

In the mild cases the head is usually large, the fontanelles and sutures open, the face small. The intelligence remains good. From time to time, for periods of several days, the child suffers from disturbances of digestion and nutrition, a febrile movement, fits of crying, restlessness, and drowsiness.

In these mild cases the disease is not of itself fatal, but the children are apt to be carried off by some of the diseases of childhood.

In the severe cases there is marked impairment of nutrition; the children are dull and stupid, irritable and drowsy; there are often strabismus, nystagmus, dilated pupils, protruded eyeballs, blindness, deafness, general convulsions, and paraplegia. There may be a febrile movement. These symptoms are interrupted by periods of improvement, but the course of the disease is usu-

ally downward, and the children die in an attack of convulsions, or of inanition, or from some intercurrent disease. Rarely they grow up to adult life, but remain more or less idiotic.

*The treatment* is mainly directed to the general condition of the patient. Careful feeding, a clean skin, good air, the administration of iron and of cod-liver oil are the essentials. During the exacerbations of the symptoms the iodide of potash may be of service.

4. CHRONIC HYDROCEPHALUS IN OLDER CHILDREN.—In older children and in adolescents we meet with cases of chronic hydrocephalus, not congenital, but occurring after meningitis, after injuries, and without discoverable cause.

In some cases of acute meningitis, the patient, after going through the symptoms of this disease and beginning to improve, makes only an attempt at convalescence, again becomes worse, is alternately delirious and stupid, has a moderate febrile movement, and emaciates. After continuing in this condition for weeks, he may recover or die.

In the cases which occur after injury and without discoverable cause, the clinical history is like that of the chronic hydrocephalus of young children, but without the enlargement of the head.

## THE PHARYNX AND TONSILS.

THE nose, the pharynx, and the mouth are lined with a continuous tract of mucous membrane, which, on account of its situation, exposed to a great variety of irritants, is very often the seat of acute and chronic inflammations. The chronic inflammations are best treated by the specialist; the acute inflammations are usually left in the hands of the general practitioner.

Throughout this whole tract the structure of the mucous membrane is essentially the same—an outer covering of epithelium, a vascular stroma, a large supply of mucous glands.

On each side of the pharynx is a lymphatic gland of some size—the tonsil. The shape of the tonsils is peculiar; it is that of a flat mass of lymphatic glandular tissue folded on itself so as to form a wrinkled ovoid body, of which the outer surface is covered with a layer of epithelium. The wrinkles are the crypts of the tonsils.

We will consider :

Acute catarrhal pharyngitis.

Acute tonsillitis.

Acute croupous tonsillitis.

Suppurative tonsillitis.

### ACUTE CATARRHAL PHARYNGITIS.

This is a simple, acute, exudative inflammation which involves circumscribed portions of the pharynx, or the entire throat with the tonsils, or extends also into the nose.

*Lesions.*—The mucous membrane is congested and swollen. The function of the mucous glands is at first arrested, so that the surface of the mucous membrane is dry; after some hours or days the function of these glands is stimulated. There is then an increased quantity of mucus, which is thin and easily dis-

charged from the surface of the mucous membrane, or thick and tenacious, so that it adheres to it.

*Causes.*—Some individuals, and the members of some families, are especially liable to acute pharyngitis; this liability is most marked in young people. The inhalation of irritating gases, or of irritating substances floating in the air, is a frequent cause of pharyngitis. Prolonged exposure to cold and wet and disturbances of the stomach also seem to be efficient causes.

*Symptoms.*—The symptoms are constitutional and local. The principal constitutional symptom is a rise of temperature, with the accompanying chills, prostration, vomiting, headache, and pains in the limbs. This fever often precedes the sore throat, and disappears before the latter has subsided.

The local symptoms are the characteristic appearance of the throat and the pain, which is increased by any movement of the muscles of the pharynx. The pain is most annoying when the inflammation involves the pillars of the fauces, the upper surface of the soft palate, and the uvula.

The cases vary as to the height of the temperature and the severity of the constitutional symptoms. In the worst cases the temperature reaches 104° F., and the patients look seriously ill.

The inflammation is a self-limited one, lasting for from four to seven days, but occasionally it continues for a longer time.

*Treatment.*—As the inflammation is a self-limited one, and naturally terminates in recovery, treatment is directed to shortening its duration and making the patient more comfortable.

The best local application at the beginning of the disease seems to be cocaine; later the mild astringents are of service.

The milder cases require nothing but the local treatment. In the more severe cases a variety of drugs are given: opium in small doses, calomel or the sulphate of magnesia in small doses, tincture of aconite in drop doses, salicylate of soda in doses of ten to twenty grains every three hours, or salol in the same doses.

Profuse sweating of the entire skin may be of service.

#### ACUTE TONSILLITIS.

*Synonyms.*—Follicular tonsillitis, ulcerative tonsillitis, spotted sore throat.

*Lesions.*—There is a simple exudative inflammation of one or both tonsils. These bodies are swollen so that they can be seen

projecting into the throat, and are congested. The crypts are filled with little white masses of mucus and epithelium, which contrast with the congested tonsils. There are no real ulcers.

The causes, symptoms, and treatment of acute tonsillitis are the same as those of acute pharyngitis.

#### CROUPOUS TONSILLITIS.

*Synonym.*—Diphtheritic sore throat.

*Lesions.*—The tonsils are congested and swollen. Their surfaces are partly or completely covered with patches of false membrane. The false membrane is composed of fibrin, pus, and necrotic epithelium. The stroma of the mucous membrane is infiltrated with fibrin and pus. The lymphatic glands in the neck may be swollen.

In bad cases there is gangrene of portions of the tonsils and of the tissues of the neck.

Streptococci and staphylococci are found in the inflamed tissues and the false membrane.

*Causes.*—This form of tonsillitis is due to local infection with streptococci and staphylococci. It is a complication of many of the infectious diseases, but especially of scarlet fever. Less frequently it occurs by itself.

*Symptoms.*—A croupous tonsillitis gives the same symptoms as does a simple tonsillitis, but much more severe.

The temperature is higher, the prostration greater, the appearance of the inflamed tonsils is quite different. The inflammation regularly runs its course within a week, but the prostration left after the disease is more marked and lasts longer.

The gangrenous forms, however, are attended with septic symptoms, and are usually fatal.

*Treatment.*—The most efficient treatment is the frequent and thorough local application of large quantities of weak solutions of bichloride of mercury, or peroxide of hydrogen.

#### SUPPURATIVE TONSILLITIS.

*Synonym.*—Quinsy sore throat.

*Lesions.*—There is in one or both tonsils an acute exudative inflammation with necrosis of tissue, which goes on to the formation of an abscess in the substance of the tonsil. A catarrhal pharyngitis accompanies the tonsillitis.

*Causes.*—There is a decided predisposition in some persons and in some families to this form of tonsillitis. Apparently, the exciting causes are the same as for simple tonsillitis, with the addition of the bacteria of suppuration.

*Symptoms.*—There is a febrile movement which precedes and accompanies the tonsillitis. Accompanying the fever are chills, headache, vomiting, pains in the back, and prostration.

The inflamed tonsil becomes gradually more and more swollen, it fills up the pharynx, pushes the soft palate forward, and projects outward into the neck. From the beginning there is a good deal of pain in the throat, and, as the swelling increases, the pain is greater, the mouth and throat are constantly filled with mucus and saliva. The patients can hardly swallow anything, they feel as if they might suffocate, they are unable to sleep. If both tonsils are inflamed and the uvula swollen, or if there is œdema of the glottis, there is real danger of suffocation. All the symptoms continue and increase until the abscess breaks and the pus is discharged. Then there is an abrupt change for the better, and in a few days the patient is well.

The inflammation regularly runs its course and terminates in rupture of the abscess within a week. If both tonsils are inflamed successively the disease lasts longer.

The patients almost uniformly recover, but it is possible for death to be produced by œdema of the glottis.

*Treatment.*—During the first twenty-four hours of the tonsillitis we may try to abort the inflammation by the use of calomel, sulphate of magnesia, aconite, salicylate of soda, salol, or cold applied to the neck.

After the abscess has been formed, the inhalation of hot steam and the application of moist heat to the neck are of service. The excessive production of mucus can be partly controlled by astringent washes and the administration of belladonna. The pain and distress can be alleviated by local applications of cocaine and the internal use of opium.

Swelling of the uvula and œdema of the glottis demand free scarification.



## THE LARYNX.

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### LARYNGISMUS STRIDULUS.

THIS name is given to two different forms of spasmodic contraction of the muscles of the larynx.

1. There is a spasmodic closure of the glottis, which is almost complete, lasts for a few seconds, and is then followed by a loud, stridulous inspiration.

This occurs in young children, usually under two years of age, who are badly nourished, or who suffer from rickets.

It occurs in attacks, which are repeated after short or long intervals.

The child suddenly stops breathing, the face and lips become pale, or slightly livid, the appearance is almost that of a dead person. Then, after a few seconds, there is a long, stridulous inspiration, and the attack is over. Sometimes the attacks are accompanied by rigidity of the muscles, or by general convulsions, or by unconsciousness. Occasionally the attacks are fatal.

At the time of an attack, if it is an alarming one, we may give inhalations of nitrite of amyl, or pass a tube through the larynx.

To prevent the attacks the nutrition of the child is to be improved in every possible way.

2. There is a spasmodic partial closure of the glottis, attended with difficult and stridulous breathing, which lasts for several hours.

This occurs in young children of all kinds, whether their previous health has been good or not. There is a decided predisposition in families and in individuals. Some of the attacks are due to indigestion, for others no cause can be discovered.

The attacks usually begin in the night and last until the next day. The child wakes up in the night with laryngeal dyspnoea, which after a time is followed by more or less venous congestion of the skin. But there is no fever, and, except for the dyspnoea,

the child does not look ill. Although the dyspnœa may appear alarming, it always eventually subsides, even if it is left to itself ; but an emetic will cause it to disappear more rapidly. The best emetics are those which act the quickest ; the yellow sulphate of mercury, apomorphia, ipecac, and antimony are those ordinarily used.

#### ACUTE CATARRHAL LARYNGITIS.

*Lesions.*—The mucous membrane of the larynx is congested, and at first dry. The stage of dryness lasts for from twelve to forty-eight hours, and is then succeeded by an increased production of mucus. With the production of the mucus the congestion and swelling of the mucous membrane diminish. The inflammation may extend to the trachea and bronchi or to the pharynx. It occurs both in adults and children.

1. *In Adults. Causes.*—The inflammation occurs without discoverable cause, after exposure to cold, from the inhalation of smoke or steam, with syphilis, phthisis, measles, scarlatina, variola, erysipelas, typhus and typhoid fevers.

*Symptoms.*—In the more severe cases there is a febrile movement, in the milder cases this is absent.

The patients have a laryngeal cough, at first dry, afterward with mucous expectoration. The voice is husky, or stridulous, or reduced to a whisper. There is more or less discomfort or pain in the larynx. In some of the cases there is laryngeal dyspnœa, continuous, but with exacerbations. The most alarming feature about this dyspnœa is that occasionally the patients stop breathing suddenly and die.

The inflammation usually runs its course within two weeks, but it may be protracted for a much longer period.

*Treatment.*—During the acute stages of the laryngitis the continuous application of hot sponges to the neck, and the inhalation of steam, are of decided service. The internal administration of tartarized antimony, or of the iodide of potash, may also be useful. When the dyspnœa is urgent the patients are to be constantly watched, in order that intubation or tracheotomy may be performed, if necessary. If the inflammation is prolonged the mineral acids, quinine, iron, and change of climate may hasten the subsidence of the disease. The local application of astringent sprays is of much service.

2. *In Children.*—The catarrhal laryngitis of children is often

called catarrhal croup. It is one of the most common diseases of childhood.

It occurs often without discoverable cause, sometimes after exposure to cold and wet, from the inhalation of steam and smoke, and as a complication of measles and scarlatina.

The disease is most common in children between the ages of one and five years, less frequent in older children. There is a well-marked predisposition to the disease in some children and in some families.

The changes in the mucous membrane of the larynx are the same as those observed in adults, but the smaller size of the larynx in children causes the swollen mucous membrane to be a still greater obstruction to the act of breathing.

*Symptoms.*—The local symptoms are : Dyspnœa, which is continuous, but with exacerbations ; in some cases attacks of laryngismus stridulus ; stridulous voice or loss of voice ; and stridulous cough.

The general symptom is a rise of temperature with its accompanying disturbances. The fever may precede, or follow, or be simultaneous with, the local symptoms.

The invasion of the disease is often sudden, and then usually occurs in the night, with the continuous dyspnœa, or an attack of laryngismus stridulus as the first symptom.

In other cases the invasion of the disease is gradual, with a croupy cough as the first symptom.

The ordinary cases last for three days and nights, the symptoms worse during the night and better during the day.

The patients vary as to the height of the temperature and the degree of the dyspnœa. Most of the patients are decidedly better by the fourth day, the improvement beginning with the production of mucus from the inflamed membranes. Occasionally, however, the laryngitis is protracted for one or two weeks, or it may be succeeded by a bronchitis.

*The Prognosis* is good, even the severe cases are seldom fatal.

*Treatment.*—For the laryngitis we apply moist heat to the neck, and give internally five- or ten-drop doses of the wine of antimony every one or two hours. For the attacks of laryngismus stridulus we give emetics. For the fever it may be proper to use small doses of antifebrin or tincture of aconite ; for the restlessness, small doses of opium. In the protracted cases small doses of calomel may be of service.

## CROUPOUS LARYNGITIS.

*Synonym.*—Membranous croup.

*Lesions.*—The mucous membrane of the larynx is congested, swollen, and infiltrated with fibrin and pus. Its free surface is coated with a false membrane composed of fibrin, pus, and necrotic epithelium.

Streptococci and staphylococci are found in the false membrane and the inflamed tissues, except in the cases in which the laryngitis is caused by a local irritant.

*Causes.*—Children are more liable to the disease than are adults. Most of the cases are examples of a streptococcus inflammation complicating measles and scarlet fever. But it may also occur in the same way with the other infectious diseases and by itself.

A similar inflammation may be excited by the inhalation of irritative vapors, such as hot steam or smoke, and by swallowing irritating fluids which find their way into the larynx.

*The Symptoms* are the same as those of a catarrhal laryngitis, but are more severe, more continuous, and last for a longer time. The temperature is higher, the dyspnœa is more severe, and the evidences of imperfect aëration of the blood are more marked. In the favorable cases, after from four to seven days the inflammation subsides, the false membrane becomes loosened, is coughed up, and the dyspnœa is relieved. In the unfavorable cases the dyspnœa continues and causes the death of the patient.

*The Prognosis* is unfavorable, especially in children under three years of age.

*The Treatment* is the same as that for a catarrhal laryngitis, but in addition it is often necessary to employ intubation or tracheotomy to relieve the dyspnœa. The inhalation of the fumes of calomel may be of great service.

## THE PLEURA.

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IN the study and treatment of the diseases of the pleura and of the lungs we are very much assisted by percussion and auscultation, by the aid of which we determine the so-called physical signs.

### THE PHYSICAL SIGNS OF THE PLEURA AND OF THE LUNGS.

**PERCUSSION.**—When we percuss the wall of the chest we obtain certain sounds, and of these sounds we note the quality, the pitch, the intensity, and the duration.

1. *Pulmonary Resonance.*—This is characterized by pulmonary quality, low pitch, considerable duration, and variable intensity. It is heard over the healthy lung.

2. *Dulness.*—Of this the quality is imperfectly pulmonary, the pitch is higher, the duration is short, the intensity is small. It is heard over the normal lung where the chest-wall is thickened by bone or muscle, and where the liver and heart are in contact with the lung. It is heard over pleuritic adhesions, over small quantities of fluid in the pleural cavities, over consolidations of the lung, emphysema, enlargements of the heart, liver, and spleen, aneurisms of the aorta, abscesses, and tumors.

3. *Flatness.*—The quality is flat, the pitch is high, the duration is short, the intensity is small. It is heard over the liver, where it is uncovered by the lung, over thick pleuritic adhesions, fluid in the pleural cavities, complete consolidation of the lung, aneurisms, abscesses, tumors, and rarely over a lung which is not consolidated, but of which the bronchi are completely obstructed.

4. *Tympanitic Resonance.*—The quality is tympanitic, the pitch is high or low, the duration is considerable, the intensity is marked. It is heard over the stomach and intestines, over air in the pleural cavities, over cavities in the lungs, over solidified and compressed lung, and with emphysema.

5. *The Cracked Pot Sound*.—The quality is metallic, the pitch is high, the duration and intensity are not very great. It is heard over cavities, and over consolidated and compressed lung.

6. *Amphoric Resonance*.—The quality is amphoric, the pitch is low, the duration and intensity are considerable. It is heard over air in the pleural cavity, over large cavities in the lung, and occasionally over consolidations of the lower lobe of the left lung.

THE BREATHING.—In listening to the breathing we distinguish the sound of inspiration and that of expiration, and of each we note the quality, the pitch, the intensity, and the duration.

1. *Pulmonary, or Vesicular Breathing*.—Of inspiration, the quality is pulmonary, the pitch is low, the duration is considerable, the intensity is variable. Of expiration, the pitch is low and the duration is shorter.

2. *Bronchial Breathing*.—Of inspiration, the quality is bronchial, the pitch is high, the intensity and duration are variable. Of expiration, the pitch is higher and the duration is longer than those of inspiration. It is heard over consolidated and compressed lung, and over cavities.

3. *Broncho-vesicular Breathing*.—This is of a character intermediate between that of vesicular and that of bronchial breathing. It is heard over the normal lung in the interscapular region, and over the lesser degrees of consolidation and compression of the lung.

4. *Cavernous Breathing*.—Of the inspiration, the quality is cavernous, the pitch is low. The expiration is longer and lower-pitched than the inspiration. It is heard over cavities and over consolidated or compressed lung.

5. *Amphoric Breathing*.—The quality is amphoric, the pitch is low, the expiration is prolonged. It is heard over large cavities in the lungs, and over pleural cavities which contain air, with perforation of the lung.

6. *Sibilant Breathing*.—The quality is sibilant, the pitch is high, the intensity is great, the expiration is prolonged.

*Sonorous Breathing*.—The quality is sonorous, the pitch is low, the intensity is great, the expiration is prolonged. Both these forms of breathing are produced by a contraction of the calibre of the larger and medium-sized bronchi. The ordinary causes of such a contraction are bronchitis and spasmodic asthma.

A special form of sibilant and sonorous breathing is pro-

duced by narrowing of the lumen of the trachea, or of the large bronchi.

**RÂLES.**—These are abnormal sounds which accompany the breathing and are not heard over the healthy lung.

1. *The Crepitant Rôle.*—This is a very fine, dry, crackling sound. It is heard at the end of inspiration, is produced in puffs, and seems to be close to the ear. It is heard with dry pleurisy, with pneumonia, and with phthisis.

2. *The Subcrepitant Rôle.*—This is a fine, moist sound ; heard with inspiration, or with expiration, or with both. It accompanies dry pleurisy, bronchitis, pneumonia, phthisis, and œdema of the lungs.

3. *The Coarse, or Mucous, Râles.*—These are loud, moist sounds, heard both with inspiration and expiration. They are heard with pleurisy, with bronchitis, with pneumonia, and with phthisis.

4. *The Gurgling Râles.*—These are loud, coarse, moist sounds, of gurgling quality. They are heard over small cavities, over compressed lung, and occasionally over pleuritic adhesions.

**FRICTION SOUNDS.**—These are produced by the rubbing of opposed surfaces of pleura coated with fibrin, or by the movement of pleuritic adhesions.

They have the characters of the crepitant, the subcrepitant, the mucous, or the gurgling rôle ; the sound is a grazing, or rubbing, or creaking one.

**THE VOICE.**—*The Pulmonary Voice.*—The quality is pulmonary, the pitch is low, the intensity and the thrill differ with the individual.

*Increased Vocal Resonance.*—The quality is pulmonary, the pitch is higher, the intensity and the thrill are greater. It is heard over the normal lung in the right infra-clavicular and in both the interscapular regions. It is also heard over consolidated or compressed lung, over cavities, and over lung which is adherent to the wall of the chest.

*Diminished Vocal Resonance.*—The intensity and the thrill are diminished, the quality and the pitch are the same as with the pulmonary voice. It is heard over small effusions in the pleural cavities, over thick pleuritic adhesions, over consolidated lung, and over lungs of which the bronchi are occluded.

*Suppressed Vocal Resonance.*—There is absence of the voice-sound with fluid in the pleural cavities, with intra-thoracic

tumors, with consolidation of the lung, and with obstructions of the bronchi.

*Bronchophony*.—The quality is bronchial, the pitch is high, the intensity and the thrill are variable. It is heard over consolidated and compressed lung, and over cavities.

*Egophony*.—This is a form of bronchophony characterized by its peculiar, shrill quality. It is heard over lung compressed by fluid in the pleural cavity, at the level of the fluid.

*Pectorilgy*.—Not only the sound of the voice is heard, but the articulation of words can be distinguished. It is heard over large cavities, and sometimes over consolidated lung.

### PLEURISY.

The pleura is a connective-tissue membrane composed of fibrillated connective tissue, with its basement substance and cells, and covered over its free surface by a layer of endothelial cells. Imbedded in it are nerves, blood-vessels, and lymphatics. According to its distribution, we speak of the pulmonary, costal, mediastinal, and diaphragmatic pleura.

The pleura may become inflamed in several different ways, and we describe :

Pleurisy with the production of fibrin.

Pleurisy with the production of fibrin and serum.

Pleurisy with the production of fibrin, serum, and pus.

Pleurisy with adhesions.

Tubercular pleurisy.

In pleurisy with the production of fibrin alone, or of both fibrin and serum, the morbid changes in the pleura are essentially the same, differing only in the presence or absence of the serum.

The first change in the pleura is simply a congestion and a falling off of the endothelial cells. Then the pleura loses its smooth, shining appearance, and looks dull and rough, this change being due to the presence of small knobs and threads of coagulated fibrin on its surface. At the same time, if serum is to be present, it begins to collect in the pleural cavity.

Next there is a swelling of, and a new growth from, the connective-tissue cells of the pleura, and an emigration of white blood-cells from the vessels. Then we find the pleura coated with a layer of fibrin in which are entangled pus-cells and new



connective-tissue cells. After this the new connective-tissue cells entangled in the fibrin become more numerous, a basement substance and new blood-vessels are formed.

Finally, the fibrin and the serum are absorbed and disappear, and the pleura is left thickened and with connective-tissue adhesions binding together its opposed surfaces.

#### PLEURISY WITH THE EXUDATION OF FIBRIN. DRY PLEURISY.

*Lesions.*—The inflammation begins in the costal or pulmonary pleura, according to the cause producing it. It extends regularly to the portion of pleura opposite to it. Usually only a circumscribed portion of the costal, pulmonary, mediastinal, or diaphragmatic pleura is involved, but sometimes the entire pleura of one side of the chest is inflamed. The inflamed pleura is coated with fibrin, and bands of fibrin form adhesions between the opposed pleural surfaces.

In rare cases the quantity of fibrin is so great as to compress the lung.

When the inflammation has subsided, the fibrin is absorbed, but permanent connective-tissue thickening and adhesions are left.

*Causes.*—Exposure to cold and wet, wounds of the chest-walls, inflammations of the lung, peritonitis, the infectious diseases, and Bright's disease are regular causes of pleurisy. In some cases there seems to be an individual predisposition to pleurisy, and the same person suffers from several attacks.

*Symptoms.*—The most constant physical sign is a friction sound—a crepitant, subcrepitant, or mucous râle, or a rubbing sound. This is heard over the inflamed portion of the pleura. It cannot be heard if only the mediastinal or diaphragmatic pleura is inflamed. It is heard only with inspiration, or with expiration also. It is usually not continuous, but requires a forced inspiration to develop it.

In some cases there is also dulness over the inflamed portion of the pleura.

In the mild cases the only constitutional symptom is pain over the inflamed pleura.

In the more severe cases there is a febrile movement attended with prostration and headache, shallow breathing, and a dry cough. These symptoms only last for a few days.

The exceptionally severe cases, with very large exudations of fibrin, resemble cases of lobar pneumonia.

The ordinary cases recover after a short time, but the patient is left with permanent thickenings and adhesions of the pleura. Such adhesions may give no further trouble, except for occasional pain; or they may form the starting-point for a chronic pleurisy with adhesions, followed by interstitial pneumonia and chronic bronchitis.

*Treatment.*—For the pleurisy we employ wet, or dry, cups, or blisters over the inflamed pleura. For the pain we use opium. While the febrile movement is present the patients should be kept in bed. So long as the friction sound persists the patient must be kept in the house if the weather is cold, but in warm weather this is not necessary.

#### PLEURISY WITH THE EXUDATION OF FIBRIN AND SERUM. PLEURISY WITH EFFUSION.

The inflammation involves the greater part of the pleura on one side of the chest. Occasionally both sides of the chest are inflamed, and when this is the case the pericardium is apt also to be inflamed. The pleural cavity contains more or less clear, or turbid, serum. The surface of the pleura is coated with fibrin, and bands of fibrin join together its opposite surfaces. The lung is more or less compressed, according to the quantity of fluid.

After the inflammation has subsided the serum and fibrin are absorbed, and thickenings and adhesions of connective tissue are left.

The compressed lung expands partially or completely. According to the expansion of the lung, there is left more or less retraction of the affected side of the chest.

*The Causes* are the same as those which produce dry pleurisy.

*Symptoms.*—The disease may run an acute or a subacute course.

1. *The Acute Form.*—The symptoms begin abruptly with chills, fever, full and frequent pulse, pains in the head and limbs, vomiting, and prostration. The breathing is frequent and shallow, there may be a dry cough, there is severe pain. The pain is referred to the inflamed pleura, or to some point in the back, or in the abdomen, or even to the opposite side of the chest. It

usually becomes less severe with the accumulation of serum in the pleural cavity.

After a few days the acute symptoms subside. The inflammatory products remain in the chest for some time longer. They may then be absorbed, or the pleurisy may take on the subacute form and last for a long time.

2. *The Subacute Form.*—The symptoms are developed gradually and slowly. The patients complain of pain in the side, of dyspnœa on exertion, of a dry cough, of loss of appetite, flesh, and strength, and they become anæmic. They are, for a time, not confined to bed, and often continue at their work. They have a little fever, the temperature normal in the morning, but running up to  $100^{\circ}$  in the afternoon.

In some cases, however, the temperature runs higher:  $100^{\circ}$  in the morning, and  $101^{\circ}$  to  $103^{\circ}$  in the afternoon. With these higher temperatures the patients lose flesh and strength more rapidly, and may sweat at night.

*The Physical Signs.*—At the beginning of the inflammation, when the pleura is coated with fibrin and but little serum has been exuded, there is a friction sound, which is a rubbing sound, or a crepitant or subcrepitant râle. After the fluid has been absorbed there is again a friction sound—a subcrepitant or coarse râle, or a creaking sound.

When a considerable quantity of fluid has accumulated in the pleural cavity there are physical signs due to the presence of the fluid and the compression of the lung.

Below the level of the fluid there is flatness on percussion, absence of voice, of breathing, and of vocal fremitus. The fluid accumulates in the lower part of the pleural cavity, compressing the lung upward and against the vertebral column; or in the posterior part of the pleural cavity, compressing the lung against the anterior wall of the chest; or it may be shut in by adhesions. The compression of the lung is in proportion to the quantity of the fluid.

At the level of the fluid there is dulness on percussion and ægophony.

Above the level of the fluid, over the compressed lung, the percussion resonance is pulmonary, or exaggerated and high-pitched, or tympanitic. The breathing is pulmonary, or exaggerated, or broncho-vesicular, or bronchial.

The affected side measures more than the opposite side of the

chest, the diaphragm is pushed down, the intercostal spaces may be forced outward, the heart may be displaced toward the opposite side of the chest.

As the fluid is absorbed the voice and breathing can be heard lower and lower down, the flatness disappears, but dulness on percussion remains for some time after all the fluid has been absorbed.

*Exceptional Physical Signs.*—The vocal fremitus may not be lost below the level of the fluid. Bronchophony alone, or both bronchial voice and breathing, may be heard below the level of the fluid, especially if the quantity of fluid is large and the lung much compressed.

Above the level of the fluid, over the compressed lung, there may be cavernous breathing and gurgling râles.

The sacculated effusions give very irregular physical signs, varying with the position of the fluid.

The acute cases of pleurisy with effusion may last for only a few weeks, but more frequently, whether acute or subacute, they last for weeks or months.

In a few cases the disease terminates fatally. Death is then often sudden, and seems to be due to congestion and œdema of the other lung, or to interference with the action of the heart.

In a moderate number of cases the inflammation changes its character, pus is added to the other inflammatory products, and the patients have empyema.

Most of the patients recover, but with a damaged pleura, and with a lung which expands more or less incompletely. In many of them the only subsequent inconvenience is some pain on the affected side of the chest; but in others there is marked retraction of the wall of the chest, chronic pleurisy with adhesions, interstitial pneumonia, or chronic bronchitis. It may also happen that such a pleurisy will be succeeded by chronic phthisis.

The displaced heart may return to its natural position, or it may remain fastened in its new place by adhesions, or it may be drawn over to the retracted side of the chest.

*Diagnosis.*—Pleurisy with effusion may be mistaken for empyema, tubercular pleurisy, pneumonia, phthisis, abscess of the liver, or tumors of the pleura. In many cases we are warranted in drawing off fluid from the pleural cavity with a fine needle, in order to establish the diagnosis.

*Treatment.*—Of the acute form of pleurisy with effusion, the

treatment is that of an acute exudative inflammation. Wet or dry cups, or blisters, over the affected side of the chest, calomel, and the sulphate of magnesia given internally, are the most efficient remedies. The patients are to be kept in bed and on a fluid diet. The pain and restlessness may be relieved by opium combined with aconite or veratrum viride, or by chloral hydrate with one of the bromides.

In subacute pleurisy with effusion we have to treat the inflammation and the accumulation of fluid within the pleural cavity. The only direct means of treating the inflammation is the use of counter-irritation over the affected side of the chest. The indirect means are keeping the patients in bed, or getting them out of doors in a suitable climate, according to the case; the use of iron, quinine, and the mineral acids; and the regulation of the diet.

If the quantity of fluid in the pleural cavity is moderate it can be removed by diuretics—the iodide or acetate of potash, the diuretic pill, chloride of sodium, caffeine, convallaria, digitalis. At the same time the urine is to be measured every day, and the ingestion of fluids somewhat restricted.

If the chest is distended with fluid this must be removed by the aspirator. In doing this the strictest cleanliness must be observed, and only a moderate quantity of the serum withdrawn. Immediately after the aspiration the use of diuretics should be begun.

#### PLEURISY WITH THE PRODUCTION OF SERUM, FIBRIN, AND PUS. EMPHYEMA.

*Lesions.*—The inflammation regularly involves the whole of the pleura on one side of the chest, less frequently a circumscribed portion of the pleura. The inflammation follows two different forms:

1. The pleura is coated with fibrin and pus, and its cavity contains purulent serum, but the pleura itself is but little changed. This form is most common in children.

2. The pleura is coated with fibrin and pus, its cavity contains purulent serum, and in addition the pleura itself is much changed. It is split up by great numbers of new cells, so that it resembles granulation tissue.

In either case the fluid accumulates in the lower part of the pleural cavity, pushing the lung upward and toward the verte-

bral column ; or in the posterior part of the pleural cavity, pushing the lung forward ; or it is sacculated in any part of the pleural cavity. The lung is usually much compressed. In old cases the pleura becomes much thickened, and may be infiltrated with the salts of lime.

The suppurative process may extend from the pulmonary pleura to the lung, and the pus then escape at intervals from the bronchi ; or it may extend from the costal pleura to the wall of the chest, and the pus escape externally.

In a few cases the inflammatory products and the superficial layers of the pleura become gangrenous.

The micro-organisms regularly found are either streptococci or pneumococci. There seems to be no special difference in the clinical symptoms whether the infection is effected by one or the other of these organisms.

*Symptoms.*—1. The inflammation may be primary, after exposure to cold, or without discoverable cause. The patients are suddenly attacked with chills, a high temperature, marked prostration, headache, pains in the back and limbs, pain over the inflamed pleura, shallow and painful breathing, sometimes cough. The symptoms may continue acutely and the patients die in a short time, or they may subside and the inflammation pass into the chronic condition.

2. The inflammation may be secondary to a pleurisy with effusion, or to a lobar pneumonia. A pleurisy with effusion may change suddenly or slowly into an empyema. The patients lose flesh and strength more rapidly, and have higher temperatures. A lobar pneumonia may run its course, convalescence be established and continue for several days, and then the temperature goes up, and there are the physical signs of fluid in the pleural cavity.

3. An empyema, after running its course for a shorter or longer time, will suddenly change, the inflammatory products become gangrenous, the patients pass into the pyæmic condition and die in a few days.

4. Abscesses in the wall of the thorax, in the liver, in the abdominal cavity, or in the lung, may rupture into the pleural cavity and set up a purulent inflammation.

The physical signs of empyema are the same as those of pleurisy with effusion, but sacculatation of the fluid and irregular physical signs are more common.

*The Course of the Disease.*—Some of the acute cases continue without any abatement of the symptoms, and terminate fatally within a short time. More frequently the course of the disease is chronic. The patients go on for months or years with fever, gradual loss of flesh and strength, and dyspnœa and cough. In some the lung is perforated and the pus from time to time coughed up through the bronchi; in some the wall of the chest is perforated and the pus imperfectly evacuated; in some there is septic poisoning.

Very rarely does spontaneous recovery take place; somewhat more frequently there is partial recovery, with absorption of some of the pus and sacculation of the remainder. Most of the patients, if not cured by proper treatment, die exhausted by the disease, or with pulmonary phthisis, or with waxy viscera.

The most difficult cases to make out are those with a sacculated empyema. The patients have more or less fever and go on week after week not getting well. The physical signs are variable and deceptive. Rather a favorite seat of such an empyema is at the root of the lung.

*The Prognosis* is more favorable in children than in adults; in those operated on early than in those operated on later. It is unfavorable after septic poisoning has begun, and when the empyema is caused by the rupture of an abscess into the pleural cavity.

*The Diagnosis* is between empyema, pleurisy with effusion, lobar pneumonia, broncho-pneumonia, tubercular pneumonia, and abscess of the liver.

*The Treatment.*—In children the disease can be cured by aspiration; but if after two or three aspirations the improvement is not decided, it is better to open the chest.

In the smaller sacculated collections of pus in adults a cure can often be effected by aspiration.

In the ordinary cases of empyema in adults the rule is to open the chest as soon as the diagnosis is made.

The regular procedure is to feel for the first rib below the angle of the scapula, to cut down on this rib and remove it up to its cartilage, to put in a large drainage-tube, sew up the wound, and dress with bichloride. The dressings are to be changed as seldom as possible, the chest is not to be washed out, and the drainage-tube should be removed at the end of the fourth week.

## CHRONIC PLEURISY WITH ADHESIONS.

*Lesions.*—There is a chronic inflammation of the pleura with the production of new connective tissue, but without fibrin, serum, or pus. The inflammation begins at some part of the pleura, and then extends until first one lung and then both are completely covered with adhesions and fastened to the wall of the chest. It is not to be confounded with the old adhesions found over so many lungs after death, but is a chronic inflammatory process with the progressive formation of more and more adhesions.

*Causes.*—The disease usually originates in the adhesions which have been left behind by previous attacks of dry pleurisy, pleurisy with effusion, or pneumonia; but in some cases no history of such previous acute attacks can be obtained.

*The Symptoms* vary with the extent of the lesions.

In the early stages the only symptoms are occasional pain over the affected part of the chest, dulness on percussion, and friction sounds.

When the disease is farther advanced, the pain continues, there is a dry cough, the breathing is imperfect, there is dyspnoea on exertion, and the area of dulness on percussion and of the friction sounds is larger.

In the advanced cases the difficulty in breathing becomes very marked, the cough is more troublesome, the heart is diminished in size and sometimes displaced, the circulation is feeble, the patients lose flesh and strength. They usually die from some intercurrent disease, but occasionally the pleurisy is the only discoverable cause of death.

*Treatment.*—The patients should live as much as possible in the open air. They may be benefited by the use of cod-liver oil, iron, quinine, or the mineral acids. They should practice daily the filling and emptying of the lungs with air in as complete a manner as possible.

## TUBERCULAR PLEURISY.

Apart from the tubercular inflammation of the pleura, which accompanies general tuberculosis and chronic phthisis, we find tubercular pleurisy occurring as a localized tubercular inflammation.

*Lesions.*—The inflammation involves regularly the whole of



the pleura on one side of the chest, the costal pleura being principally involved. The pleura is of a bright-red color mottled with small white points, or is only thickened and coated with fibrin. The tissue of the pleura is split up by the growth of new connective-tissue cells, and contains numerous tubercle granula. There is a large quantity of fluid in the pleural cavity, which is blood-stained, or purulent, or clear.

*Symptoms.*—The clinical history is that of pleurisy with effusion, or of empyema, but the patients do badly. They lose flesh and strength, the fluid accumulates rapidly after it has been drawn off, the inflammation of the pleura persists, and the patients die, either suddenly or exhausted by the disease, within a few weeks or months.

*Treatment.*—It would seem, from our experience of the treatment of tubercular peritonitis, that in tubercular pleurisy it would be good practice to open the chest.

#### HYDRO-PNEUMOTHORAX.

This name is used to designate the presence of both air and fluid in the pleural cavity. Such a condition may be established in several different ways.

There may be a gangrenous empyema with the formation of gas in the pleural cavity.

There may be an empyema with an opening through the wall of the chest or into the lung.

There may be abscesses or gangrene of the lung, perforating the pulmonary pleura.

There may be pulmonary phthisis, with softening of cheesy nodules and perforation of the pleura.

*Symptoms.*—The only cases of hydro-pneumothorax which have a special clinical history are those due to the rupture of abscesses or phthisical nodules in the lung. There is first the history of the previous lung disease. Then, suddenly, at the time of the perforation, there is severe pain, a feeling as if something had given way within the chest, urgent dyspnœa, a rapid and feeble heart action, and great prostration. The patients may die in collapse within a few hours of the commencement of the attack; or the urgent symptoms may subside, and the patients continue to live for some time with the symptoms of empyema and phthisis.

*Physical Signs.*—The affected side of the chest is larger than the other, and moves but little with respiration. The heart and the diaphragm are displaced. Vocal fremitus is absent. Percussion gives, above the level of the fluid, exaggerated pulmonary or tympanitic resonance, or flatness ; below the level of the fluid, flatness. Auscultation gives, above the fluid amphoric breathing, or absence of breathing ; below the level of the fluid, absence of breathing. If the patient is shaken, we get the splashing sound of the fluid in the chest called “succussion.” We may also get the sound resembling drops of liquid falling into liquid, called “the metallic tinkle.”

## THE LUNGS.

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THE lungs first appear as two small protrusions on the front of the œsophagus. They are diverticula of the hypoblast surrounded by mesoblast. The formative process consists in the budding of hypoblastic into mesoblastic substance; the hypoblast furnishing the lining epithelium and the mesoblast the stroma. As the lungs continue to develop they look like glands with acini and ducts.

Just before birth the lungs are fully formed, but the air-spaces are not dilated and are completely lined with cuboidal epithelium. After birth the air-vesicles are dilated and their epithelium is flattened.

As the child becomes older the air-spaces occupy a larger, and the bronchi and the stroma a smaller, part of the lung.

In the adult lung the stroma is arranged so as to divide the lung up into lobules, six or seven-sided blocks, each side 5 to 15 mm. long. But few of the lobules are entirely separated from each other by the stroma. The small bronchi enter the lobules irregularly and break up into terminal bronchioles. The bronchioles terminate in the air-passages. The air-passages are tubular spaces of irregular shape with air-vesicles surrounding and opening into them on every side. Some air-vesicles, however, are given off directly from the bronchioles. The air-passages constitute the larger part of the lung, their walls have the same structure as those of the air-vesicles, their function in the act of breathing is the same as that of the vesicles.

The walls of the air passages and of the air-vesicles are composed of a thin connective tissue membrane, reinforced by elastic fibres, with an abundant system of capillary vessels. These walls are no longer covered, as at birth, with a continuous layer of epithelium. Only a few scattered cells of epithelial type are left as indications of the fœtal epithelium.

The important portions of the lung are :

1. Its stroma, which holds together all the component parts of the lung, and, as the pulmonary pleura, invests its external surface. In the stroma are imbedded blood-vessels, lymphatics, and nerves. The stroma may be the seat of exudative, purulent, or productive inflammation.

2. The bronchi, of which the walls are composed of connective tissue, muscle, and cartilage, and are lined with a mucous membrane. The bronchi constitute the larger part of the lungs during fœtal life ; in the young child they continue to predominate, but in the adult they occupy a relatively smaller space. They may be the seat of acute catarrhal, of chronic catarrhal, of croupous and of productive inflammation.

3. The air-passages and air-vesicles, by which the actual breathing and blood-aërating function of the lungs is performed. The structure of the walls of the air-spaces in the adult is that of a simple connective-tissue membrane which may be attacked by exudative or productive inflammation.

4. The blood-vessels, which are large and numerous and form a network of capillaries in the walls of the air-spaces. Any obstruction of the blood-vessels interferes with the breathing function of the lung, and, if long continued, causes changes in its structure.

5. The lymphatics—vessels throughout the lung and large nodes at the root of the lung. Inflammation of the bronchi is regularly attended with inflammation of the lymph-nodes, especially in children.

The stroma, the bronchi, and the air-spaces may be inflamed separately or together.

#### BRONCHITIS.

*Causes.*—Inflammation of the bronchi occurs at all ages. The acute form is more common in children, the chronic form is more common with emphysema, with heart disease, and in old persons.

Persons living in cities, those who are much confined to the house, and those whose health has been enfeebled are especially liable to the disease.

There is in some persons a very well-marked predisposition to inflammation of the bronchi.

The disease is especially prevalent in cold and damp climates, and during the cold and wet months of the year. During some years it is much more prevalent than during others.

As exciting causes of bronchitis we recognize exposure to cold, the inhalation of irritating gases and substances, and of pathogenic bacteria.

Measles, whooping-cough, influenza, and many of the infectious diseases are often complicated by bronchitis.

### 1. THE ACUTE CATARRHAL BRONCHITIS OF ADULTS.

*Definition.*—An acute exudative inflammation of the mucous membrane of the bronchi.

*Lesions.*—The inflammation involves the trachea, the larger bronchi and the medium-sized bronchi, not as often the smaller ones. As a rule the bronchi of both lungs are equally inflamed. The mucous membrane of the bronchi is congested and swollen, at first it is dry, afterward coated by an increased production of mucus. There are also desquamation of the epithelial cells, emigration of white blood-cells, diapedesis of red blood-cells. The lumen of the bronchi may be narrowed either by the congestion and swelling of the mucous membrane or by the contraction of the muscular coat.

*Symptoms.*—The mild cases of acute bronchitis are attended with cough, accompanied by scanty mucous sputa, by pain over the sternum, and a moderate feeling of indisposition. There may be no physical signs, or a few coarse râles, or sibilant and sonorous breathing. The natural duration of the inflammation seems to be about a week, but it is often prolonged for a much longer time.

In the more severe cases there is a troublesome cough with mucous or muco-purulent sputa, often streaked with blood. The quantity of expectoration is sometimes very large—more than a pint in twenty-four hours.

The febrile movement precedes and accompanies the cough. It is usually not over  $101^{\circ}$ , but there are cases with temperatures of  $103^{\circ}$  to  $104^{\circ}$  throughout the disease.

In some patients bronchial asthma is the most prominent and distressing symptom.

The physical signs are coarse râles and sibilant and sonorous breathing. As a rule these sounds are equally distributed over

both lungs, but occasionally they are confined to one side of the chest.

The cases vary as to their severity and duration. The patients are sometimes very ill, but do not often die. The ordinary duration of the disease is two weeks, but it may last much longer.

In some of the cases a localized broncho-pneumonia is developed, with dulness and increased vocal resonance over the consolidated portion of the lung.

There are cases in which the bronchitis continues for weeks and months. The cough and muco-purulent expectoration continue. There is an irregular fever with evening exacerbations and sweating at night. The patients lose flesh and strength, and sometimes look very badly. In these cases the sputum should always be examined for tubercle bacilli.

*Treatment.*—The milder cases are not confined to bed, they can eat solid food, but are to be cautioned against undue exposure. Many of them recover without treatment, but in some remedial measures are necessary.

The severe cases have to be kept in bed, and mainly on a fluid diet.

For the cases of bronchitis which need treatment we employ :

(1) Counter-irritation. The most efficient counter irritation is by dry cupping over the entire chest. More moderate counter-irritation can be effected by large mustard plasters, irritating liniments, and poultices frequently renewed.

(2) Drugs. The specific drug for acute bronchitis is ipecac. It seems to be most efficient when given frequently in small doses—gr.  $\frac{1}{10}$ th every hour. There may be an advantage in combining the ipecac with other drugs: extr. belladonna, gr.  $\frac{1}{40}$ ; pulv. Doveri, gr.  $\frac{1}{10}$ ; pulv. ipecac, gr.  $\frac{1}{20}$ ; quiniæ sulph., gr.  $\frac{1}{4}$ , made into a pill or tablet, taken every hour.

When there is a large expectoration of mucus with a good deal of pus mixed with it, 10- to 20-drop doses of tr. of nux vomica are of much service.

The bronchial asthma seems most frequently to be due to contraction of the walls of the bronchi. If this is the case, nitrite of amyl, nitro-glycerine, chloral hydrate, or opium are indicated.

Sometimes, however, the asthma is associated with congested, swollen, but dry bronchi, then is indicated the use of muriate of

pilocarpine, gr.  $\frac{1}{16}$ ; fl. extr. quebracho, ℥ x.; fl. extr. grindelia robusta, ℥ x., or larger doses of ipecac.

In the protracted cases there is much advantage in sending the patient to a dry inland climate.

Of useful drugs for chronic bronchitis there is a considerable number, sometimes one sometimes another proving the most serviceable: the mineral acids, the preparations of turpentine, iodide of potash, strychnia, and inhalations of creosote.

## 2. THE ACUTE CATARRHAL BRONCHITIS OF OLD PERSONS.

*The lesions* are the same as in the bronchitis of adults.

*The symptoms* are more severe in proportion to the extent of the bronchitis, and the disease is not as well borne as it is in adults. There are, from the first, much prostration; an irregular febrile movement; a rapid and feeble pulse; difficult and oppressed breathing; cough with mucous expectoration; restlessness, sleeplessness, sometimes delirium; loss of appetite, nausea, vomiting. There are coarse and subcrepitant râles over both lungs, or only over a portion of one lung. In some cases no râles can be heard.

An acute bronchitis in old persons is often alarming and sometimes fatal.

*The treatment* is the same as in adults; but the nursing is even more important, the difficulty in feeding the patients is greater, and stimulants are more likely to be necessary.

## 3. THE ACUTE CATARRHAL BRONCHITIS OF CHILDREN.

*The lesions* are the same as in adults: congestion and swelling of the mucous membrane, after a time an increased production of mucus with some subsidence of the swelling. But as the lungs of children are smaller, as the bronchi constitute a relatively larger portion of the lung, as the bronchi are smaller and more easily occluded, as such an occlusion may even be complete with unaëration of portions of the lung, so we find bronchitis in children to interfere seriously with the function of breathing and to constitute a serious disease.

*Symptoms.*—An acute bronchitis may be preceded by coryza, pharyngitis, tonsillitis, or laryngitis; it may occur as a primary inflammation; or it may complicate measles, whooping-cough, or one of the infectious diseases.

In the mild cases there is no fever, the children hardly feel sick ; but they cough, and coarse râles can be heard over both lungs. The inflammation regularly runs its course and subsides within one or two weeks.

In the majority of these mild cases no treatment is necessary, not even for the cough. It is wise, however, to keep these patients in the house until the bronchitis has subsided, unless the weather is warm and good.

In the more severe cases the invasion of the inflammation may be marked by general convulsions. There is a well-marked febrile movement, the temperature higher in the afternoon, sometimes falling even to the normal in the morning. The pulse is rapid, but usually not feeble. The breathing is rapid, sometimes insufficient ; it may be made worse for a time by distention of the stomach with food. There may be alternating restlessness and drowsiness. There are subcrepitant and coarse râles heard over both lungs, sometimes early, sometimes late in the disease.

The inflammation regularly runs its course within two weeks, and the patients recover. But they are often alarmingly ill for several days, and may die from the disease.

In infants only a few weeks old, rapid breathing, fever, and prostration are the only symptoms, and the disease proves fatal within a few days.

*Treatment.*—During the first days of the inflammation we employ counter-irritation over the chest by turpentine, croton-oil, or poultices, and give small doses of calomel or sulphate of magnesia. Later ipecac, aconite, and opium may be of service. But it is to be remembered that in young children all drugs may do harm, and that too little treatment is better than too much.

#### 4. CHRONIC CATARRHAL BRONCHITIS.

Chronic catarrhal bronchitis may from the first have the character of a chronic inflammation ; it may follow one or more attacks of acute bronchitis ; it may complicate gout, emphysema, chronic endocarditis, interstitial pneumonia, pleuritic adhesions, phthisis ; it may be produced by the inhalation of irritating substances.

*Lesions.*—The mucous glands produce too much mucus, they may be hypertrophied ; the walls of the bronchi are thickened



or thinned ; the lumen of the bronchi may be narrowed or dilated.

*Symptoms.*—In the mild cases the patients are only troubled by the cough and expectoration, while their general health remains good.

In the severer cases the cough is more troublesome, the expectoration more profuse. There may be constant or spasmodic dyspnœa. There is an irregular fever, with loss of flesh and strength. There are coarse and subcrepitant râles, sometimes sibilant and sonorous breathing.

The disease runs a protracted course, better in the summer and worse in the winter. The patients are more likely to die from some intercurrent disease than from the bronchitis.

*The Treatment* is the same as that for the protracted form of acute bronchitis.

#### PNEUMONIA.

The terms pneumonia or pneumonitis are employed to designate the inflammations of the parenchyma of the lung as distinguished from those of the bronchi and the pleura. By the parenchyma of the lung we mean the air-vesicles, the air-passages, and the smallest bronchi.

There are a number of different forms of pneumonia, distinguished from each other by their causes, their lesions, and their symptoms. In the present state of our knowledge we cannot make a scientific classification of these, but have to be contented to describe the different varieties of pneumonia under arbitrary names.

We distinguish, therefore :

Primary lobar pneumonia.

Secondary lobar pneumonia.

Lobar pneumonia, with the formation of new connective tissue.

Broncho-pneumonia.

Pneumonia of heart disease.

Interstitial pneumonia.

Tubercular pneumonia.

Syphilitic pneumonia.

## PRIMARY LOBAR PNEUMONIA.

*Definition.*—An infectious inflammation, with exudation from the blood-vessels and the growth of pathogenic bacteria, which involves principally the air-spaces of the lungs.

*Synonyms.*—Croupous pneumonia, fibrinous pneumonia, lung fever, pneumonitis.

*Etiology.*—Lobar pneumonia is a very widely distributed disease. There are few countries in which it does not prevail, the mortality ranging from 1.10 to 2.30 per cent. for each 1,000 inhabitants.

In the United States the disease is of more frequent occurrence in the South than in the North. This, the following table, based on the eighth and ninth census reports, conclusively shows:

STATES WHOLLY OR IN GREAT PART ABOVE THE 39TH PARALLEL.			STATES WHOLLY OR IN GREAT PART BELOW THE 39TH PARALLEL.		
	Per 1,000 Deaths.	Per 1,000 Inhab- itants.		Per 1,000 Deaths.	Per 1,000 Inhab- itants.
1. Maine .....	51.22	0.62	1. Delaware .....	56.41	0.68
2. New Hampshire .....	62.46	0.96	2. Maryland .....	59.59	0.70
3. Vermont .....	58.58	0.59	3. Virginia .....	75.66	0.94
4. Massachusetts .....	56.29	0.98	West Virginia .....		
5. Rhode Island .....	58.23	0.78	4. North Carolina .....	71.34	0.80
6. Connecticut .....	55.96	0.72	5. South Carolina .....	102.58	1.26
7. New York .....	60.55	0.87	6. Georgia .....	99.54	1.17
8. New Jersey .....	51.61	0.59	7. Florida .....	113.55	1.39
9. Pennsylvania .....	44.84	0.58	8. Alabama .....	123.81	1.47
10. Ohio .....	60.27	0.65	9. Mississippi .....	127.21	1.68
11. Indiana .....	80.66	0.88	10. Louisiana .....	94.15	1.75
12. Illinois .....	77.94	0.96	11. Texas .....	105.43	1.58
13. Michigan .....	69.64	0.67	12. Arkansas .....	183.42	2.98
14. Wisconsin .....	54.55	0.50	13. Kansas .....	112.13	1.49
15. Minnesota .....	55.30	0.39	14. Kentucky .....	78.49	0.95
16. Iowa .....	75.32	0.61	15. Tennessee .....	84.03	1.04
17. Nebraska .....	87.30	0.93	16. Missouri .....	103.96	1.41
18. Oregon .....	55.76	0.34	17. Nevada .....	81.30	1.18
19. Colorado .....	50.67	0.48	18. California .....	46.77	0.65
			19. District of Columbia .....	60.87	0.98
Average .....	61.43	0.69	Average .....	93.70	1.27
			Excess over Northern States ..	32.27	0.58

In most countries in the temperate zone the maximum frequency of the disease is from February to May.

As regards New York City, I compiled from the records of the Board of Health the deaths from pneumonia from March 1, 1871, to March 1, 1875, 7,873 cases. Nearly half the entire num-

ber was in children under five years of age. The smallest mortality was in persons from ten to twenty years old.

The majority of the cases occurred in March, April, and May, December, January, and February, the minority in June, July, and August.

In persons over five years old the curves of mortality are very regular, and the difference between the spring and winter months and the rest of the year very striking. In persons over seventy years of age the same law prevails. In children under five years of age the curves are much less regular.

The curves of mortality in general correspond with those of temperature, the greatest mortality with the lowest temperature and the greatest daily range of temperature.

The disease may occur in epidemics, confined to prisons, barracks, asylums, or involving certain districts. There seems to be no question that persons living an out-of-door life in the country are less liable to the disease than are persons living in cities.

It has always been a matter of importance to determine whether pneumonia is contagious, whether a person suffering from the disease can communicate it to others. The disease certainly occurs in circumscribed local epidemics and from time to time we see several persons in one house successively attacked. On the other hand it is well known that physicians, nurses, and relations, who take care of cases of pneumonia, are not often attacked by the disease. For the present the question must be considered an unsettled one.

For the production of a lobar pneumonia there must be a cause of inflammation, such as exposure to cold, and the growth of pathogenic bacteria. The organism most frequently found is the bacillus described by Fraenkel; it is said to be found in over ninety per cent. of all the cases. This same bacillus is also found with pleurisy, pericarditis, peritonitis, and cerebro-spinal meningitis, and is regularly present in the saliva and nasal secretions of healthy persons. Much less frequently the bacillus of Friedländer, or other streptococci of suppuration are found.

The old conception of pneumonia was that it was simply an inflammation of the lung. Within a few years the opinion that, on the contrary, it is a general disease, of which the inflammation of the lung is the characteristic lesion, gained very general acceptance. With our present knowledge it seems most probable that

pneumonia belongs to the class of infectious inflammations. That is, it is an inflammation of the lung accompanied by the growth of pathogenic bacteria. The growth of these bacteria is attended with the formation of poisonous chemical products, and according to the quantity and virulence of these products the symptoms of general poisoning are more or less marked.

When exposed to the same exciting causes children under five years of age usually have broncho-pneumonia; children between the ages of five and fifteen have either broncho-pneumonia or lobar pneumonia; adults usually have lobar pneumonia.

*Morbid Anatomy.*—The inflammation regularly involves the whole of one lobe, or the whole of one lung, or portions of both lungs.

Juergensen, from a study of 6,666 cases, gives the following table to show the relative frequency of the situation of the lesion :

	Per cent.
Right Lung.....	53.70
“ “ upper lobe.....	12.15
“ “ middle lobe.....	1.77
“ “ lower lobe.....	22.14
“ “ whole lung.....	9.35
Left Lung.....	38.23
“ “ upper lobe.....	6.96
“ “ lower lobe.....	22.73
“ “ whole lung.....	8.54
Both Lungs.....	8.07
“ “ both upper lobes.....	1.09
“ “ both lower lobes.....	3.34

The inflammation in acute lobar pneumonia is of pure exudative type, characterized by congestion, emigration of white blood-cells, diapedesis of red blood-cells, and exudation of blood-plasma, and formation of fibrin, while the tissue of the lung remains unchanged. For clinical purposes it is important to have as distinct an idea as possible of the condition of the lung while it is the seat of such an exudative inflammation, so that we describe the condition in which the lung is found while the inflammation is going through its regular stages of congestion, exudation, and resolution.

During the first hours of the inflammation only irregular

portions of the lobe which is to be inflamed are involved; later, the entire lobe. The lung is congested, œdematous, tough, but not consolidated. The air-spaces contain granular matter, fibrin, pus-cells, red blood-cells, and epithelial-cells. The epithelium remaining on the walls of the air-spaces is swollen; there are large numbers of white blood-cells in the capillaries. The larger bronchi are congested, dry, or coated with mucus; the small bronchi contain the same inflammatory products as do the air-spaces. The pulmonary pleura, as a rule, is not coated with fibrin. This is called the stage of "congestion." The stage of congestion regularly only lasts a few hours, but it may be protracted for several days.

When the exudation of the inflammatory products has reached its full development, the presence of these products within the air-spaces and bronchi causes the lung to be solid, and at this time the lung is said to be in the condition of "red hepatization." The lung is now consolidated, red, its cut section looks granular, the granules corresponding to the plugs of inflammatory matter within the air-spaces. For some time after death the inflammatory products remain solid, and the cut section of the lung dry, but later, with the commencement of post-mortem changes, these products soften and the cut section is covered with a grumous fluid. The air-vesicles, the air-passages, the small bronchi, and sometimes the large bronchi, are filled, and distended with fibrin, pus-cells, red blood-cells, and epithelium. In spite of the pressure on the walls of the air-spaces, the blood-vessels in their walls remain pervious. The pulmonary pleura is coated with fibrin and the interstitial connective tissue of the lung is infiltrated with fibrin. The hepatized lobe is increased in size, sometimes so much so as to compress the rest of the lung. About one-fourth of the fatal cases die in the stage of red hepatization, at any time from twenty-four hours to eleven days after the initial chill.

After the air-spaces have become completely filled with the exudation, if the patient continues to live, there follows a period during which the exudate becomes first decolorized, and then degenerated. This is the period of "gray hepatization." The lung remains solid, its color changes, first to a mottled red and gray, then to a uniform gray. The coloring matter is discharged from the red blood-cells and the exudate begins to degenerate and soften. The lung is found passing from red to gray hepatic.

zation at any time between the second and the eighteenth day of the disease. It is found completely gray at any time from the fourth to the twenty-fifth day. About one-half of the cases die in the condition of mottled red and gray hepatization; about one-fourth in the condition of gray hepatization.

If the patients recover the exudate undergoes still further degeneration and softening, and is removed by the lymphatics. This is the stage of "resolution." It should commence immediately after defervescence and be completed within a few days. But it may not begin until a number of days after defervescence, or it may be unusually protracted.

*Modifications of the Inflammation.*—The lung, instead of being freed from the exudate at the regular time, may remain in the condition of gray hepatization for weeks.

The quantity of inflammatory products may be so great that the blood-vessels are compressed and portions of the lung become necrotic.

There may be an excessive production of pus-cells, with infiltration of the walls of the air-spaces and of the stroma of the lung.

The bronchitis may be developed in an unusual degree, and involve not only the bronchi of the inflamed lung but also those of the other lung.

The pleurisy may be unduly developed at any time in the course of the pneumonia, or after it has subsided.

In the lobar pneumonia which accompanies epidemic influenza there is often an intense catarrhal bronchitis with a large production of muco-pus; and in some cases an excessive congestion of the lung with comparatively little hepatization.

The lymphatic vessels in the pulmonary pleura and in the septa between the lobules may be filled with pus cells, and the pleura and the septa infiltrated with fibrin and pus.

It is often stated that lobar pneumonia can be changed into a tubercular pneumonia, or a chronic pneumonia, but I believe that the cases thus described are really examples of pneumonias which were of tubercular, or of productive character from the very outset.

*Symptoms.*—Physical signs. During the stage of congestion the lung is more dense, but is not consolidated, the bronchi and some of the air-spaces contain inflammatory products, the pleura is not yet coated with fibrin. The percussion-note, therefore, remains unchanged, or its pitch becomes higher, its duration

shorter, and its quality less distinctly pulmonary. The respiratory murmur is either rude, or diminished in intensity. The inflammatory products in the small bronchi may give a subcrepitant râle. If the larger bronchi are also inflamed, there may be coarse râles and sibilant and sonorous breathing. As there is no fibrin yet on the pleura, there is no crepitant râle. It is evident, therefore, that during this stage of a pneumonia we must expect that the physical signs will either not be very marked, or else absent altogether.

During the stages of red and gray hepatization the air-spaces and small bronchi are filled with inflammatory products and impervious to air. The larger bronchi are coated with mucus or filled with fibrin. But although in all cases the lung is consolidated there is a good deal of difference as to the quantity of inflammatory products, the size of the consolidated lobe, the closeness with which its surface is applied to the chest-wall, and the degree of motion of which it is capable. The pulmonary pleura is coated with fibrin, occasionally there is serum in the pleural cavity.

The percussion sound, therefore, is more or less dull or flat, or tympanitic, or of cracked-pot quality.

Any considerable quantity of fluid in the pleural cavity gives flatness.

A lobe of which the air-spaces are distended with exudation so that the lobe is increased in size, and its surface pressed closely against the wall of the chest gives either marked dulness; or flatness; or tympanitic resonance; or, in young persons, the cracked-pot sound.

The dulness on percussion is less marked, or is even absent altogether, if the inflamed lobe is very much congested but contains little exudation; if the quantity of exudation is not sufficient to distend the air-spaces and increase the size of the lobe; if the area of consolidation is small, or is situated in the central portions of the lung; or if the ribs have undergone the senile changes which cause them to give increased resonance. It is especially in old persons that these reasons for the absence of dulness on percussion often exist.

The vocal fremitus is regularly increased over the consolidated lung. There is no satisfactory reason why this should not always be the case, but as exceptional conditions we find the vocal fremitus diminished, or absent.

Bronchial voice and breathing should be heard over the consolidated lung ; but we may get bronchial voice without bronchial breathing, or we may get neither bronchial voice nor breathing.

The absence of bronchial voice seems to be due sometimes to the incomplete consolidation, sometimes to the occlusion, of the large bronchi. The absence of bronchial breathing may be due to occlusion of the large bronchi, or to the absence of movements of the lung. As the crepitant râle is due to the friction of the fibrin on the surface of the pleura, the râle will not be produced unless fibrin is present, and the lung capable of movement. So we find in different cases a great difference as to the presence or absence of the crepitant râle. In some cases we only get it after coughing, or with a forced inspiration, in some cases it is only heard at the beginning of red hepatization while the lung still moves, in some cases it is heard throughout the stages of red and gray hepatization, in some cases it is absent altogether.

In the stage of resolution the products of inflammation are softened and rapidly absorbed, the air re-enters the small bronchi and air spaces, the lung moves more and more freely. So with the increased motion of the lung we get the crepitant râle due to the friction of the pleura. With the softening of the inflammatory products we get the subcrepitant and coarse râles in the bronchi. The bronchial voice and breathing disappear, if they have been present. Normal vesicular breathing becomes more and more distinct. The percussion-note loses its dull, or flat, or tympanitic, or cracked-pot quality, and approaches nearer and nearer to the normal, but yet the changes in the percussion-note last the longest of all the physical signs, and even long after complete resolution some dulness is often present.

It is always to be remembered that it is in old persons that the physical signs are the least constant and the least well marked.

*Rational Symptoms.*—In from one-sixth to one-third of the cases there are prodromic symptoms. Chilliness, a little fever, general malaise and feelings of oppression about the chest continue for from one to four days. These symptoms probably correspond to a protracted period of congestion.

In about ninety per cent. of the cases there are, during the first twenty-four hours, one or more decided chills, and it is from the time of the chill that we count the days of the disease.



The temperature rises at once and reaches its maximum by the afternoon of the first, second, or third day, but very often the highest temperature of the disease will be reached during the twenty-four hours preceding defervescence. An afternoon temperature of  $104^{\circ}$  F. and a morning temperature of  $102^{\circ}$  or  $103^{\circ}$  are about the normal temperatures of a lobar pneumonia. A sudden rise of temperature during the course of the disease indicates the extension of the pneumonia, or the development of a complication. But the removal of the patient from one house to another is regularly followed by a rise of temperature, and in persons not suffering from malarial poisoning and not taking antipyretics there are quite often irregular rises and falls of several degrees of temperature which we cannot account for. Pneumonias involving the upper lobes usually have high temperatures. The height of the temperature is usually in proportion to the severity of the disease, but patients may get worse with a falling temperature, or may die with temperatures below the normal, or, very rarely, have no rise of temperature throughout the disease.

Defervescence may take place at any time from the second to the eighteenth day of the disease. It occurs most frequently on the seventh day, next on the fifth, eighth, sixth, and ninth days in order. The fall of temperature usually begins in the evening, and, within from six to forty-eight hours it reaches the normal, or for a time falls below it. Occasionally a rapid defervescence with a fall of temperature to  $97^{\circ}$  or  $96^{\circ}$  is attended with so much prostration and such a feeble heart-action that the condition of the patient is alarming. It is said that epistaxis, hæmaturia, or hemorrhage from the bowels may accompany defervescence.

In the pneumonias which complicate epidemic influenza there may be no marked defervescence, but a gradual fall of temperature extending over many days, and in some cases the fever persists after the pneumonia has resolved.

A rise of temperature after several days of partial or complete defervescence usually means pleurisy or empyema, but it may indicate a fresh pneumonia, abscess of the lung, or gangrene of the lung.

The condition of the heart's action and of the pulse are of great importance. In a favorable case the pulse ought to be about 100 to the minute and fairly full. A pulse of over 120 is always cause for anxiety. The liability to failure of the heart's action, either gradual or sudden, constitutes one of the greatest

dangers of a pneumonia. It is not certain what the cause of the heart failure is, but it seems probable that it is due to the effects of the poison produced by the pathogenic bacteria of the disease. In persons already suffering from chronic endocarditis failure of the heart's action is exceedingly dangerous.

The breathing during the invasion of the disease is rapid and oppressed. As the disease goes on the character of the breathing varies with the severity of the case. Rapid, labored, and insufficient breathing indicates either inflammation of a large part of the lung, excessive congestion of the lung, an intense general bronchitis, failure of the heart's action, or an intense pleurisy and pericarditis.

Cough may be developed as one of the first symptoms, or come on at any time in the disease, or be deferred until resolution has commenced. In old persons the cough is often slight, or absent altogether.

The characteristic sputa of pneumonia are little rounded, viscid pellets of red, yellow, or brownish color, mixed with thin fluid mucus, the so-called rusty sputa. In rare cases, with a severe invasion, the patients may cough up a little pure blood at the beginning of the disease. In the bad cases the sputa are changed and the patients cough up considerable quantities of a thin, dark-colored fluid. In some cases throughout the disease there will be nothing but a little white mucus coughed up from time to time. In cases with an excessive catarrhal bronchitis the quantity of expectoration may be very large and like that of an ordinary bronchitis. It is by no means unusual, especially in old people, for the expectoration to be absent altogether.

Pain over the inflamed lung, referred to the region below the nipple, is developed within twelve hours after the initial chill in the majority of cases, and after three or four days gradually disappears. This pain is sometimes so intense as to be for a time the principal symptom. But in some persons there is no pain until resolution commences and the cough becomes troublesome. In old persons there is often not only an absence of pain, but there are no abnormal sensations whatever in the chest.

The expression of the face is characteristic. There is a deep red flush at about the centre of each cheek, and the expression of the face is a curious mixture of anxiety and apathy.

The skin may be either hot and dry, or bathed in perspiration throughout the disease.

The tongue is coated with a white fur, but in the favorable cases remains moist. A dry tongue indicates a severe form of the disease.

Headache, restlessness, and sleeplessness are troublesome during the first days of the disease in many of the patients. Delirium and stupor belong to the severe cases. The alcoholic patients often have an active delirium, or delirium tremens. Old persons often become apathetic, or mildly delirious.

The invasion of the disease is often attended with vomiting ; less frequently with diarrhœa.

The urine is diminished in quantity and high colored. It often contains a little albumin and a few casts, due to acute degeneration of the kidney. It is said that sodium chloride and some of the other inorganic salts are diminished in quantity.

*Complications.*—At any time in the course of a pneumonia, or after defervescence, there may be developed pleurisy with effusion, or empyema on the same side as the pneumonia. The ordinary course is for the patient to get to the sixth or seventh day of his pneumonia and behave as if he were about to get well, but yet without complete defervescence. After a few days the temperature rises a little with pleurisy with effusion, a good deal with empyema. The physical signs are those of fluid in the pleural cavity, but very often bronchial voice and breathing are heard below the level of the fluid.

A catarrhal bronchitis involving the larger bronchi of both lungs is not infrequent, especially with the pneumonia of epidemic influenza. The patients cough up large quantities of mucus, often mixed with blood. Coarse râles and sibilant and sonorous breathing can often be heard over both lungs. The dyspnœa is more troublesome, the temperature higher, and the tendency to heart failure and venous congestion more decided.

Acute pericarditis is a serious complication. It may be that there will be nothing to call attention to the condition of the heart, and the case seems only to be a pneumonia of severe type. Or there may be decided precordial pain, a rapid and feeble pulse, a pericardial friction sound, rapid breathing, and cyanosis. It occasionally happens that the symptoms of the pericarditis are more marked than those of the pneumonia, so that it is even possible for the pneumonia to be overlooked.

A previously existing chronic endocarditis adds much to the dangers of pneumonia. The heart's action is likely to be dis-

turbed, and the condition of general venous congestion established. It must not be forgotten in such cases that a well-marked mitral or aortic stenosis may give no murmur at all.

Acute meningitis is an infrequent complication, but a very fatal one. It may run its course without giving any distinctive symptoms; or the delirium may be more active, with contractions of groups of muscles, or general convulsions.

Jaundice is seen both in mild and in severe cases. It seems to be a non-obstructive jaundice without symptom.

Acute degeneration of the kidneys, of mild type, is of ordinary occurrence. It seems to do the patients no harm and to give no symptoms except the presence of albumin and casts in the urine.

Acute exudative nephritis is of much less frequent occurrence. It is not likely to prove fatal of itself, but it may add to the dangers of the pneumonia.

Persons already suffering from chronic nephritis are very unfavorable subjects for an attack of pneumonia; not many of them recover. Quite often the chronic nephritis is one which has given no symptoms until the time of the pneumonia.

*The Course of the Disease.*—1. The regular cases. These cases may be mild or severe, they may terminate in recovery or in death, but they all have this in common, that the clinical picture is that of an inflammation of the lung with comparatively little evidence of general poisoning. The patients begin with the chills, rapid rise of temperature, sleeplessness and restlessness, vomiting, pain in the side, cough, expectoration, and dyspnœa. These symptoms continue either mildly or severely; after the third day comes the liability to heart failure, and, finally, at the regular times come defervescence or death. The severity of the symptoms is directly in proportion to the extent of lung involved and to the intensity of the inflammation.

2. The infectious cases. In these cases the symptoms have no necessary relation with the extent of lung involved, in many of them only a part of one lobe is inflamed. The patients behave as if they were poisoned. The prostration is marked, the emaciation rapid. The temperatures are high, the heart's action is rapid and feeble, the tongue is dry, the cerebral symptoms are marked, and the disease is very regularly fatal.

3. The rational symptoms run their regular course, but the physical signs are slow in developing, so that it may be as much as eight days before they are really well marked.

4. The inflammation, instead of remaining confined to the lobe in which it began, may extend to other portions of the lungs. Each extension of the inflammation is attended with an exacerbation of the symptoms.

5. There are rare cases in which nearly the whole of both lungs becomes at once inflamed, the interference with breathing is overwhelming, and death results very soon.

6. Resolution instead of beginning within one or two days after defervescence, as it should, may be delayed for from one to ten weeks. And yet, even after these long periods, the inflammatory products may be absorbed.

7. In some cases, not necessarily belonging to the infectious class, nor alcoholic, the delirium is an unusually marked symptom. In some of these patients the delirium continues for some days after defervescence, or even after resolution is completed. In a few cases the delirium is succeeded by permanent insanity.

8. Persons already suffering from chronic alcoholism, if they have an attack of lobar pneumonia, are likely to have high temperature, active delirium, or delirium tremens.

9. The pneumonia of old persons often runs an irregular course. The extent of lung inflamed may be small and the physical signs uncertain; a little dulness on percussion, a few subcrepitant râles, a diminished intensity of breathing, or even no physical signs at all. The patients usually have chilliness, or a distinct chill, to mark the invasion of the disease, and more or less fever while it is running its course. The appetite is lost, and there may be nausea and vomiting. The pulse is rapid and often feeble. Either stupor or a mild delirium are often present. The prostration is out of proportion to the extent of lung inflamed. The characteristic cough, expectoration, and pain in the chest are absent altogether, or imperfectly developed; even the breathing may not be at all changed. But the disease is very fatal in old persons, and some of them die quite suddenly after seeming to be only moderately sick for a few days.

10. The pneumonia which accompanies epidemic influenza has, in New York, presented certain peculiarities. In many cases there was an intense catarrhal inflammation of the larger bronchi of both lungs, with profuse expectoration of mucus and more or less blood. Some of the fatal cases showed very marked congestion of the inflamed lung with comparatively little consolidation, and this corresponded with an imperfect development of the

physical signs of consolidation during the patient's life. Very often there was no regular defervescence, but a slow fall of temperature extending over a number of days, and sometimes not reaching the normal until after resolution was completed. In some cases the whole duration of the disease was unusually long, and defervescence and resolution did not take place until after three or four weeks. Failure of the heart's action, with venous congestion of the lungs and other viscera was often present. The pneumonia was followed by empyema in an unusually large number of cases.

11. The course of the disease is changed by the complicating lesions in meningitis, bronchitis, pleurisy, pericarditis, endocarditis or nephritis.

*Modes of Death.*—The patients die with heart failure just before defervescence; from the extent of the inflammation; from general poisoning; from one of the complications; from thrombosis of the coronary arteries.

*Duration.*—In the cases which recover, defervescence takes place at any time from the second to the thirty-second day, most frequently on the seventh or fifth. Resolution is accomplished within a few days after this, but may be delayed up to ten weeks.

In the fatal cases death may take place at any time from five hours to thirty days—most frequently on the seventh, eighth, and tenth days.

In persons over seventy death is most common on the fifth, sixth, and seventh days.

The mortality from pneumonia is a considerable one, ranging in hospitals from twelve to forty-four per cent. It seems to be the general impression that the disease is more fatal now than it was a number of years ago, but it is difficult to determine this accurately. A study of this subject has been made by Drs. Townsend and Coolidge, who have worked up the records of the Massachusetts General Hospital from 1822 to 1889. They arrived at the following conclusions:

1. In the one thousand cases of acute lobar pneumonia treated at the Massachusetts General Hospital from 1822 to 1889, there was a mortality of twenty-five per cent.

2. The mortality has gradually increased from ten per cent. in the first decade to twenty-eight per cent. in the present decade.

3. This increase is deceptive, for the following reasons, all of which were shown to be a cause of a large mortality:

(a) The average age of the patients has been increasing from the first to the last decade.

(b) The relative number of complicated and delicate cases has increased.

(c) The relative number of intemperate cases has increased.

(d) The relative number of foreigners has increased.

4. These causes are sufficient to explain the entire rise in the mortality.

5. Treatment, which was heroic before 1850, transitional between 1850 and 1860, and expectant and sustaining since 1860, has not, therefore, influenced the mortality-rate.

6. Treatment has not influenced the duration of the disease or of its convalescence.

*Treatment.*—Lobar pneumonia is a disease for which there is no routine treatment applicable to every case, on the contrary much judgment is required to decide what is the best way of managing each patient.

In many of the simple cases the course of the disease is so regular and mild that we do not care to interfere with it at all. The patients are put to bed, are given a fluid diet and are allowed to go through the disease and get well.

On the other hand, in some of these simple cases although the disease needs no treatment there are symptoms which require our attention. The headache, restlessness, and sleeplessness may be allayed by the bromides, sulfonal, or opium. Excessive pain in the chest requires larger doses of opium, and in some patients the use of large poultices. My own belief concerning the value of poultices in pneumonia is that they are of no service except so far as they give comfort to the patients. Even in the simple cases there is often the danger of failure of the heart's action, and this danger, although it exists after the third day of the disease, seems to be at its greatest during the hours just preceding convalescence. Throughout the disease we watch the heart, the pulse, and the color of the skin and lips. As soon as the heart fails or there is venous congestion of the skin it is proper to use cardiac stimulants—whiskey, digitalis, strophanthus, or caffein. A very good combination is five grains of potassium iodide, one minim of fluid extract of digitalis and twenty minims of fluid extract of convallaria given together

every three hours. If the venous congestion is very marked a hypodermic injection of one-fiftieth grain of nitro-glycerine will often give great temporary relief.

There are many cases in which, although the course of the disease is regular, the symptoms are severe. The temperature is over 104° F., the pulse is over 120, the breathing is insufficient, venous congestion is evident. So many of these patients die that it is not easy to resign one's self to a simply expectant treatment. The plans of treatment most frequently adopted for such cases are: Venesection employed once, or repeated several times, the quantity of blood taken to be considerable. This plan is not often employed at the present time.

Large doses of calomel, 12 to 30 grains, placed dry on the tongue, from one to four such doses. This plan seems to answer well for some cases, to be of no use in others, and is attended with the risk of producing salivation.

Small doses of calomel, one-fourth to one grain given every hour up to six doses. This seems to be of moderate efficacy.

Drachm doses of magnesium sulphate given every hour up to eight doses. This again does not seem to be of much value.

Either tr. aconite or veratrum viride in doses of from two to five drops, at first every hour and later at longer intervals, have given good results in the hands of some physicians.

Cold affusions to the chest, cold baths, blisters, the antipyretic drugs, quinine, and carbonate of ammonia, have all been much used, but are all of doubtful efficacy. In the cases which behave as if the patients were poisoned, with high temperature, cerebral symptoms, dry tongue, rapid and feeble pulse, and rapid emaciation, we naturally use alcoholic and cardiac stimulants freely, but in spite of all most of these patients die.

An excessive catarrhal bronchitis may be benefited by repeated dry cupping over the entire chest, and by the administration of small doses of ipecac every hour.

For my own part, in the cases of pneumonia which require treatment, it has seemed to me, as I have watched the disease in my own practice and in that of other physicians, that:

1. We have no plan of treatment which controls the inflammation of the lung except in so far as we can diminish the venous congestion of this organ.

2. We have as yet no means by which we can prevent or con-



trol the poisoning from the chemical substances produced by the growth of the pathogenic bacteria.

3. Reduction of the temperature, while it may make the patient more comfortable, has no effect on the course of the disease.

4. The only thing that we can control with any certainty is the circulation of the blood, and, if we do control this in such a way that the proper relative quantity of blood is contained in the arteries and veins, the congestion of the lungs will be diminished, the intensity of the inflammation made less, and the danger of heart failure lessened. This means that we must keep throughout the disease, no matter how high the temperature, a pulse of between 90 to 100 to the minute, without increased tension, soft, of good quality, and of good strength.

To affect the circulation in this way we have at our command a number of drugs which increase the force of the heart's action and which dilate the arteries. It is by a combination of such drugs that we can hope to regulate the circulation in the way in which we desire.

The particular combination which has seemed to me to be the most reliable is that of aconitia,  $\frac{1}{8}$  milligr. ; digitalia,  $\frac{1}{8}$  milligr., and whiskey in doses of from one drachm to one ounce. The digitalia and aconitia are given together at intervals of from one to three hours, the whiskey every three or four hours. The guide for the frequency of the use of these drugs is the effect on the circulation. We try to keep a pulse of between 90 and 100 and of good quality ; with this the breathing will be better and the disposition to general venous congestion diminished. The temperature, however, is not affected by this plan of treatment.

#### SECONDARY LOBAR PNEUMONIA.

*Definition.*—An exudative inflammation involving one or more lobes of the lungs, occurring in persons already suffering from some disease, or injury.

*Etiology.*—Persons who are confined to bed by an infectious disease, by injuries or inflammations of the brain and spinal cord, by surgical operations, or by severe injuries, are liable to have venous congestion of the dependent portions of the lung, and to inhale substances which can irritate the lung. In this way they often contract either a true broncho-pneumonia, or a pneumonia which somewhat resembles a lobar pneumonia.

*Morbid Anatomy.*—The inflammatory process involves irregular areas of one or of both lungs. We find these areas after death in the condition of red or gray hepatization and surrounded by congested lung, but no complete consolidation of an entire lobe. The inflammation is of exudative type, with fibrin, pus, and epithelium in the air-spaces and small bronchi.

*Symptoms.*—In many cases the pneumonia can hardly be said to give either rational symptoms or physical signs. We find the lesion after death but are not certain of its existence during life. But in some cases there are chills, fever, rapid breathing, pain, cough, and expectoration, with the physical signs of bronchitis, or of consolidation of small portions of the lung.

*The Treatment* of such a pneumonia is unsatisfactory.

#### LOBAR PNEUMONIA WITH THE FORMATION OF CONNECTIVE TISSUE.

It is well known that in some forms of inflammation of the lung there is a production of new connective tissue around the bronchi and blood-vessels, in the septa between the lobules, and in the walls of the air-spaces. It is not as well known that in these same forms of pneumonia there may be also a production of new connective tissue in the cavities of the air-spaces and of the small bronchi. This new tissue either grows directly from the walls of the air-spaces, or is formed out of plugs of coagulated matter and of cells which are formed within their cavities.

Such a productive pneumonia has been recognized under a variety of names: gray induration, fibroid induration, cirrhosis, interstitial pneumonia, chronic pneumonia, desquamative pneumonia, parenchymatous pneumonia, etc. If we look over all the different lungs in which such a productive pneumonia has been developed, we find that they can be classified as follows:

1. A productive pneumonia associated with the growth of tubercle bacilli.
2. A productive pneumonia associated with the growth of actinomyces.
3. A productive pneumonia due to the inhalation of particles of coal or of stone.
4. A productive pneumonia caused by constitutional syphilis.
5. A productive pneumonia secondary to changes in the pleura.
6. Broncho-pneumonia.
7. A special form of lobar pneumonia.

It is concerning this last variety of productive pneumonia that our information is the least exact, and it is to this variety that I wish especially to call attention. The ordinary belief has been that it is possible for a regular exudative lobar pneumonia, instead of resolving, to be succeeded by a chronic productive inflammation; I believe, on the contrary, that a regular exudative lobar pneumonia terminates only in resolution or in death, and that lobar pneumonia with the production of new connective tissue is from the first a special form of inflammation of the lung. My reason for this belief is that I have seen a number of lungs which seem to show the different stages of the inflammatory process.

The literature on the subject is not very abundant. Charcot<sup>1</sup> describes this condition as following one or more attacks of ordinary lobar pneumonia. Coupland<sup>2</sup> gives a very good description with drawings, and believes that the new tissue is formed from the intra-alveolar exudation of ordinary lobar pneumonia. Kidd<sup>3</sup> describes two cases with a subacute history, which he regards as cases of lobar pneumonia terminating in induration. Buhl<sup>4</sup> considers the disease to be a primary one, which runs a subacute course and has nothing to do with ordinary pneumonia. Heitler<sup>5</sup> gives an account of the disease as observed in five cases. The development of the disease, he says, is more or less acute, with fever, dyspnoea, cough, prostration, sometimes rigors; the constitutional depression is much less marked than in acute pneumonia. The fever is irregular, and not over 102°. The sputum is mucous, muco-purulent, or fetid. In two of the cases there was retraction of the wall of the thorax. The consolidation involved in three cases the right upper lobe, in one case the whole right lung, in one case the lower right lobe. The hepatization was smooth, with necrotic and cheesy areas and cavities. The course of the disease was subacute, but with an acute invasion, lasting from fifty days to nine months and nineteen days. Wagner<sup>6</sup> has described six cases apparently belonging to this group, running a subacute course with retraction of the wall of the chest, but terminating in recovery.

<sup>1</sup> Rev. Mens. de Med. et Chir., 1878, p. 776.

<sup>2</sup> Transactions London Pathological Society, vol. xxx., p. 224.

<sup>3</sup> Lancet, April 5, 1890.

<sup>4</sup> Buhl: Briefe, p. 47.

<sup>5</sup> Wiener med. Wochenschrift, 1884 and 1886.

<sup>6</sup> Deutsch. Arch. f. klin. Med., vol. xxxiii.

I have seen twelve cases which seem to belong to this group, and to demonstrate that there is a form of lobar pneumonia which is, from the outset, anatomically distinct from the ordinary form. It is from the first an exudative inflammation with the production of new tissue, not a simple exudative inflammation. Such an inflammation naturally lasts longer, and is more likely to become chronic than is the case with a simple exudative inflammation. I can see no reason to believe that in ordinary lobar pneumonia the pus and fibrin are ever replaced by connective tissue.

The development of the lesion seems to be as follows :

1. Congestion of the lung ; exudation of serum, fibrin, and pus into some of the air-spaces ; the formation in other air-spaces of irregular plugs with prolongations from one space into others, the plugs composed of a nearly homogeneous or finely fibrillated material, none of them large enough to fill or distend the air-spaces ; a swelling and thickening of the walls of the air-spaces, with a very considerable increase in the number of epithelial cells which cover them ; more or less general catarrhal bronchitis ; fibrin on the pulmonary pleura.

2. New cells, of the type of connective-tissue cells, are formed in the plugs ; the walls of the air-spaces are more swollen, and may be infiltrated with small round cells ; new blood-vessels are formed in the plugs, which can be artificially injected from the pulmonary vessels. The gross appearance of the lung at this time is usually characteristic. One or more lobes are consolidated, they are not large, as in ordinary pneumonia, their color is red or gray, the cut surface is smooth, not granular.

3. The growth of new connective tissue within the air-spaces, in their walls, and along the arteries and bronchi, is so extensive that many of the air-spaces are obliterated. The surface of the lung is now covered with connective-tissue adhesions ; the bronchi contain muco-pus ; the lung is red, mottled with white, or gray, or black ; it is dense and hard ; portions of it may be necrotic, or cheesy, or broken down into cavities.

Four of my cases illustrate the first period of the development of the lesion :

CASE I.—The duration of the disease was ten days. The entire left lung was consolidated, small, smooth, of gray color, with fibrin coating the pulmonary pleura. The right lower lobe was

partly hepatized and red. The walls of the air-spaces were thickened and coated with epithelial cells. There was a growth of new connective tissue around the blood-vessels and bronchi. The air-spaces contained small, anastomosing plugs of a nearly homogeneous matter.

CASE II.—The duration of the disease was seventeen days. The left lower lobe was consolidated, small, red, and smooth, its surface covered with old adhesions. The walls of the air-spaces were thickened, their cavities contained the plugs already described.

CASE III.—The duration of the disease was fourteen days. There was a general bronchitis. The right upper lobe was consolidated, of reddish-gray color, its pleura coated with fibrin. The walls of the small bronchi were thickened and infiltrated with cells. The walls of the air-spaces were thickened and coated with epithelium. Some of the air spaces contained pus and fibrin, others the plugs already mentioned.

CASE IV.—The duration of the disease was seven days. The left lower lobe presented the regular picture of the red hepatization of ordinary pneumonia. The left upper lobe was consolidated, small, smooth, and red. The walls of the air spaces were thickened and coated with epithelium, their cavities contained pus and fibrin, or the plugs.

Six of my cases illustrate the second period of the development of the lesion :

CASE V.—Duration nineteen days. The right upper lobe was consolidated, smooth, and red. There was a growth of new connective tissue around the arteries, in the septa, and in the walls of the air-spaces. Some of the air-spaces contained epithelium, others plugs of the same shape and appearance as seen in the preceding group of cases, but there were, in addition, connective-tissue cells imbedded in the basement substance composing the plugs.

CASE VI.—Duration sixteen days. The right middle and lower and left lower lobes were consolidated, small, and smooth. The walls of the air spaces were thickened. Some of their cavities contained fibrin and pus, others plugs of connective tissue containing blood-vessels.

CASE VII.—Duration twenty-three days. The left lower lobe

was consolidated, small, and gray. The walls of the air spaces were thickened ; they contained plugs of connective tissue.

CASE VIII.—Duration twenty-eight days. The left upper lobe was consolidated, small, smooth, and black. The walls of the air-spaces were thickened ; they were covered with epithelium, and contained plugs of connective tissue.

CASE IX.—Duration six days. General bronchitis. The left upper lobe was consolidated, large, and red. The walls of the air-spaces were thickened. Some contained pus and fibrin, others plugs of connective tissue.

CASE X.—Duration thirteen days. The left lower lobe was in the condition of ordinary red hepatization. The right upper lobe looked like the resolution of an ordinary pneumonia, and some of the air-spaces contained degenerated exudation, but in others there were plugs of connective tissue.

Two cases illustrate the third period of development of the lesion :

CASE XI.—Duration fifty two days. The left lung was covered with old adhesions, consolidated, hard, smooth, mottled red and white, small. The growth of new connective tissue in the walls of the air-spaces and in their cavities had nearly obliterated the natural structure of the lung.

CASE XII.—Duration fifty-one days. The pleura was thickened and coated with fibrin. The left pleural cavity was half full of serum. The left upper lobe was consolidated, and of a pinkish-white color. It was almost entirely changed into connective tissue.

*Etiology.*—In three of the cases there was a distinct history of prolonged exposure to cold and wet. In one case the symptoms followed immediately after the patient having fallen into an excavation. In one case, for twenty days before the initial chill the patient was miserable, and had a troublesome cough. In one case, for twenty-one days before the initial chill, the patient suffered from headache, loss of appetite, and prostration. In one case, under observation throughout, there was an attack of lobar pneumonia terminating in resolution after eleven days ; the patient was discharged from the hospital well, and after an interval of eighteen days came the beginning of the fatal attack

in the other lobe of the same lung. In one case the patient stated that he had an attack of pneumonia five years before, and that for one year he had been troubled with cough and mucopurulent expectoration.

*Symptoms.*—In ten of the cases the invasion of the disease was marked by chills and a rapid rise of temperature. There was cough in all the cases, the sputa rusty in five cases, mucopurulent in two cases, bloody in one case. The temperature was rarely over 104° F., and in some of the cases not over 100° F. In seven of the cases delirium is noted as a prominent symptom. One case was supposed to be acute phthisis, one acute meningitis, and one acute general tuberculosis. Three of the patients passed fairly into the typhoid condition. The physical signs of the consolidation of the lung were well marked, except in one case. The duration of the disease was for 6, 7, 10, 13, 14, 16, 17, 19, 23, 28, 51, and 52 days; in most of the cases longer than that of an ordinary lobar pneumonia.

There seems to be, therefore, a form of lobar pneumonia which is anatomically different from the regular form. Its physical signs are, of course, the same, but its clinical symptoms are somewhat different. Although the patients have the same chill, fever, cough, expectoration, and pain as in the regular cases, yet there is something about the course of the disease which makes its diagnosis possible, even during its early days. The temperatures do not run high, but the tendency to cerebral symptoms and the typhoid state is very marked, and most of the cases run a protracted and subacute course. Concerning the prognosis, it is not possible to speak certainly, but there seems no reason why recovery should not be possible.

#### BRONCHO-PNEUMONIA.

*Definition.*—An infectious inflammation with exudation from the blood-vessels, a formation of new connective tissue and the growth of pathogenic bacteria, which involves principally the walls of the bronchi and the air-spaces which surround the inflamed bronchi.

*Synonyms.*—Capillary bronchitis, lobular pneumonia, catarrhal pneumonia.

There seems to be no form of pneumonia which does not have associated with it more or less bronchitis, so that every pneumonia is in one sense a broncho-pneumonia. But it has

long been recognized that there is one form of inflammation of the lung which is different from others and in which the share of the bronchi is especially important. For several reasons, however, the popular notions concerning the disease have become somewhat confused. It was seen that in some cases, while there was a bronchitis extending down to the small bronchi, there was no consolidation of the lung, and so these were called cases of capillary bronchitis. It was seen that there were cases of general bronchitis with consolidation of circumscribed portions of the lung, and it was inferred that the inflammation extended from the bronchi to the air-spaces which empty into them, so these were called cases of lobular pneumonia. This, however, was an error in observation. Areas of atelectasis do correspond to bronchi, but the areas of consolidation do not so correspond. It was seen that in some cases the symptoms and lesions could not be distinguished from those of phthisis, and it was not understood that the only real difference between the two was the presence or absence of the tubercle bacilli. A number of curious ideas were connected with the term "catarrhal inflammation" and it was not realized that a catarrhal inflammation is nothing but an exudative inflammation occurring in a mucous membrane.

It is to be regretted that the erroneous idea still exists that a broncho-pneumonia is simply an inflammation of the bronchi which extends to the air-spaces opening into these bronchi.

*Etiology.*—Broncho-pneumonia is the ordinary pneumonia of children, it is frequently seen in young persons, and occasionally in adults and old persons.

It occurs as a primary inflammation, is often secondary to measles, whooping-cough, and diphtheria, less frequently to the other infectious diseases. Persons confined to bed by injury or disease, and persons with emphysema are liable to subacute forms of broncho-pneumonia.

The disease is most frequent during the cold and wet months of the year; in some cases there is a history of exposure to cold, in others no exciting cause is discoverable.

Children crowded together in asylums are especially liable to the disease.

The same patient not infrequently suffers from several attacks of broncho-pneumonia.

The pathogenic bacteria are either the pneumococci of lobar pneumonia, or the bacteria of suppuration.



*Morbid Anatomy.*—In persons who die from broncho-pneumonia the lungs after death present considerable variety in their gross appearance. The mucous membrane of the trachea and large bronchi may appear to be normal, or is congested and coated with mucus, or the small bronchi may contain pus. The walls of the small bronchi are thickened so that a section of the lung looks studded with little nodules. In some cases most of the small bronchi of both lungs have their walls thickened in this way, more frequently it is only the bronchi of one lung or of one lobe. Around the bronchi whose walls are thickened are zones of consolidated lung from the size of a pin-head to that of a pea. Scattered through one or both lungs are irregular areas of consolidation, having no definite relation with the bronchi. They may be so large and numerous that an entire lobe or an entire lung is completely consolidated. The pulmonary pleura may be coated with fibrin. In the lungs of very young children there may be consolidated shrunken portions due to collapse of the air-vesicles, the so-called areas of atelectasis. The same condition is found with bronchitis, and in children who die so soon after birth that the whole of the lungs have not become aërated. The bronchial glands are usually swollen and inflamed.

The smaller bronchi may be dilated. In the portions of lung which are not consolidated the air-spaces may be somewhat dilated. Occasionally some of the air-spaces are ruptured, and the septa between the lobules are infiltrated with air.

In order to understand the true anatomical characters of broncho-pneumonia, it is necessary to determine which of these different changes is essential and constant and which are accessory and inconstant.

The essential and constant lesion is a productive inflammation of the walls (not the mucous membrane) of the bronchi, and of the air-spaces immediately surrounding the inflamed bronchi. The walls of the bronchi are thickened and infiltrated by a growth of new cells; the walls of the air-spaces are thickened, their cavities are filled with new connective tissue, or with fibrin, pus, and epithelium. The inflammation is from the first not exudative, but productive, that is, with the formation of new tissue. It involves the medium-sized and smaller bronchi of both lungs, but is not everywhere equally severe; in some parts of the lungs the lesions are much more marked than in others.

The accessory lesions, some of which are present in one case and some in others, are :

1. A catarrhal inflammation of the mucous membrane of the bronchi.
2. An exudative inflammation of the air-spaces, which fills their cavities with fibrin, pus, and epithelium, and produces consolidation of larger or smaller portions of the lungs. In young children the epithelial cells which line the air-spaces are much more numerous than they are in adults, so when children's lungs are inflamed the epithelial cells form a larger part of the inflammatory product than they do in the lungs of adults.
3. An exudative inflammation of the pleura, which coats it with fibrin.
4. Dilatation of the bronchi, of which the walls are the seat of productive inflammation.
5. Areas of atelectasis.
6. Simple, or tubercular inflammation of the bronchial glands.

As the inflammation of the walls of the bronchi and of the air-spaces surrounding them is from the first a productive inflammation it follows the law which governs that form of inflammation. It is apt to last for a longer time than does an exudative inflammation, and it is liable to change into a chronic productive inflammation.

It is not uncommon, therefore, for a broncho-pneumonia to continue for several weeks, or to be followed by permanent changes in the lungs.

If the broncho-pneumonia becomes chronic the inflammation of the walls of the bronchi and of the air spaces which surround them continues, we then find that the bronchi are dilated, their walls are thickened, they are surrounded by zones of connective tissue ; or part of a lobe, or an entire lobe, is entirely changed into connective tissue. The pulmonary pleura may also be very much thickened.

*Symptoms.*—In very young infants the only symptoms are : Fever, prostration, and rapid breathing. There is no cough ; there are no physical signs. The disease is almost certainly fatal within a very few days.

In older children the broncho-pneumonia may be preceded by the symptoms of measles, of whooping-cough, of coryza, of pharyngitis, or of a catarrhal bronchitis of the larger tubes ; or

it may begin without having been preceded by any other morbid conditions.

There is a good deal of difference in the different cases as to the severity of the invasion. The more severe cases are ushered in by one or more general convulsions; or by a rapid rise of temperature, vomiting, oppressed breathing, and delirium. The milder cases begin with lower temperatures, moderate prostration, and increased frequency of breathing.

After the disease is established the patients continue to have a febrile movement. The temperature in most cases is irregular, but on many days up to  $105^{\circ}$  F. Very often the temperature is of distinctly remittent type, a morning temperature of  $99^{\circ}$  to  $100^{\circ}$  F., and an evening temperature of  $104^{\circ}$  to  $105^{\circ}$  F. It is to be noticed, however, that in children both bronchitis and lobar pneumonia are also often accompanied by fever of a remittent type. It may very well be that this disposition to remission belongs rather to the age of the patient than to the character of the disease. The height of the temperature varies from day to day, sometimes with the progress or extension of the inflammation, sometimes without discoverable cause. In the cases which recover it requires several days for the temperature to fall to the normal. The height of the temperature is regularly in proportion to the severity of the broncho-pneumonia; with temperatures of over  $105^{\circ}$  F. the mortality of the disease is considerable. There are, however, patients in whom the temperature runs between  $99^{\circ}$  and  $100^{\circ}$  F. who do very badly.

In children the pulse is more rapid than it is in adults, and is also more easily rendered rapid by disease. So in broncho-pneumonias pulses of from 140 to 170 to the minute are not uncommon, and in bad cases the pulse can hardly be counted.

An increase in the frequency of the breathing is almost constantly present, as much as 40 to the minute even in mild cases, up to 60, 70, or 80 in the bad cases. It is of importance to notice not only the frequency of the breathing, but also how much air enters into the lungs. The breathing may be made worse for a time by distention of the stomach.

Sleeplessness, restlessness, and delirium are often present, and sometimes very troublesome. They seem to depend partly on the fever, partly on the interference with breathing, partly on the temperament of the child.

The face is flushed, the tongue is coated and sometimes dry,

there may be vomiting or diarrhœa ; sometimes there is pain over the chest.

Cough is often present, dependent on the catarrhal bronchitis rather than on the broncho-pneumonia. The sputa are swallowed rather than coughed up. They may collect in the stomach and be vomited up.

The urine may contain a little albumin and a few casts, the kidneys being the seat of acute degeneration.

The physical signs vary with the condition of the lung. If the inflammation is limited to the walls of the bronchi and to the air-spaces immediately surrounding them there are no physical signs. If a catarrhal bronchitis is present there are coarse and subcrepitant râles. If there is a diffuse pneumonia, with consolidation of a considerable portion of the lung, there are dulness on percussion and bronchial voice and breathing. If there is fibrin on the pleura, there are crepitant or subcrepitant râles. The signs of the consolidation and of the pleurisy are usually developed between the second and fifth days, but it is not uncommon for them to be delayed until a much later period.

The duration of broncho-pneumonia in children varies very much in different cases. Of the fatal cases the larger number die within two weeks, but some prove fatal within two days, and some are protracted for seven or eight weeks. In the cases which recover the constitutional symptoms continue for from one to three weeks in the majority of patients, but it is by no means unusual for the active symptoms to continue for six or eight weeks, and yet the patients make a perfectly good recovery. Resolution requires a longer time than in lobar pneumonia, from seven to fourteen days in most of the cases.

*The Cerebral Cases.*—In many of the cases of broncho-pneumonia there are cerebral symptoms—convulsions, restlessness, and delirium—but in some patients these symptoms are developed to such a degree, and are so out of proportion to the pulmonary symptoms, that the cases require a separate description.

The symptoms resemble those of an acute or a tubercular meningitis. They may begin and go on acutely, with high fever, prostration, one or more convulsions, alternating delirium and stupor. Or the course is more subacute, loss of appetite, vomiting, moderate prostration, not very high fever, alternating stupor and delirium. These symptoms may continue for from two to ten days before there are any pulmonary symptoms. Then, as

the pulmonary symptoms are developed, the cerebral symptoms subside.

*Persistent Cases.*—If, after the subsidence of the acute broncho-pneumonia a chronic inflammation persists, the children begin to improve, but yet do not get well.

In some the cough and the physical signs continue, the appetite is poor, the children do not gain flesh and strength, but yet they are not sick in bed—often not confined to the house.

In others the same symptoms exist, there is also an irregular fever, and the patients are sick in bed.

Of these protracted cases some recover entirely ; some recover with permanent consolidation of a portion of the lungs ; some die exhausted by the disease ; some go on to have a chronic interstitial pneumonia which lasts for many years.

In some cases of acute broncho-pneumonia, the accompanying inflammation of the bronchial glands may be of tubercular character, and this may serve at a later period as the focus of infection which causes an acute general tuberculosis.

*Treatment.*—If the inflammation is confined to the walls of the bronchi and the air-spaces around them, counter-irritation of the wall of the chest and antiphlogistic remedies are not likely to be of service. If, on the other hand, catarrhal bronchitis and general congestion of the lungs are present, with rapid and labored breathing, it may be well to use cups, or irritating liniments, or poultices over the chest, and to give repeated small doses of calomel or the sulphate of magnesia. Throughout the disease good results may be obtained from the continued use of ipecac in small doses.

The sleeplessness, restlessness, and delirium are best relieved by opium ; in children who cannot take opium we may use the bromides or asafœtida. The disposition to convulsions seems to be lessened by the use of the bromides. If the pulse is too full, with high temperatures, the children are made more comfortable by the tincture of aconite. Alcoholic stimulants are not to be used unless there is decided failure of the heart's action. The feeding and nursing of the child are of the greatest importance.

If resolution is delayed, or if the broncho-pneumonia persists, we employ iron, quinine, mineral acids, oxygen, cod-liver oil, and above all, change of air.

In adolescence the clinical picture of broncho-pneumonia is the same as it is in children, but the cerebral symptoms are not

developed to the same extent, and they are more likely to cough up blood.

In adults the disease presents itself to us under several different forms :

1. The patient has an ordinary attack of catarrhal bronchitis lasting for several days. Instead of getting well promptly, however, the patients continue to cough and to feel sick, and, on examining the chest, we find a circumscribed area where there is dulness on percussion and loud, high-pitched voice. This consolidation of the lung does not, however, last very long, and the patients make a good recovery.

2. The patients are suddenly attacked with a very severe and general broncho-pneumonia. There are chills, a rapid rise of temperature, headache, pains in the back and chest, vomiting, great prostration, a rapid pulse which soon becomes feeble, very bad breathing—rapid, labored, and insufficient—venous congestion of the skin and of the viscera, cough, at first dry, then with profuse mucous and blood-stained sputa, sleeplessness, restlessness, and delirium, and albumin in the urine. There are coarse, subcrepitant and crepitant râles over both lungs, sibilant and sonorous breathing; the percussion-note is normal, or exaggerated, or dull. The disease lasts for from seven to fourteen days; it is very apt to prove fatal.

*Treatment.*—The most efficient remedies are the energetic use of wet or dry cups over the entire chest, the administration of calomel or the sulphate of magnesia in small and repeated doses, ipecac, the inhalation of oxygen gas, and stimulants.

3. There is a form of broncho-pneumonia in adults which resembles lobar pneumonia. There is a general catarrhal bronchitis, with broncho-pneumonia and consolidation of one or more lobes. The symptoms and physical signs are like those of lobar pneumonia, but with some difference. The invasion of the disease is not as sudden, and the pulse is more rapid, the cerebral symptoms are more constant, the expectoration is like that of bronchitis, the physical signs are more slowly developed, the duration of the disease is rather longer and resolution is slower.

4. There is a form of broncho-pneumonia which resembles tubercular broncho-pneumonia. The invasion of the symptoms is gradual and the disease is protracted over a number of weeks. The patients have more or less cough and expectoration, at first mucous, later muco-purulent, but not containing tubercle bacilli.

There is a moderate fever, with evening exacerbation and sweating at night. The physical signs are those of bronchitis and of consolidation of circumscribed portions of the lung. The patients have no appetite and lose flesh and strength. Often the diagnosis depends upon the examination of the sputa. After a number of weeks in some of the patients the inflammation subsides and a complete recovery is made, but in others it continues and proves fatal.

*Treatment.*—The patients are to be kept in bed and as well fed as possible. The most efficacious treatment seems to be the continued inhalation of the vapor of creosote.

5. Persons suffering from emphysema sometimes get up a subacute broncho-pneumonia, which may prove fatal.

6. Broncho-pneumonia, especially of the lower lobes, is secondary to the infectious diseases, to injuries, to surgical operations and to any conditions which are likely to cause congestion of the lungs and the inhalation of streptococci.

#### THE PNEUMONIA OF HEART DISEASE.

*Definition.*—A chronic productive inflammation of the lungs caused by chronic congestion, and resulting in thickening of the walls of the air-spaces, filling of their cavities with epithelial cells and a deposition of pigment.

*Synonyms.*—Brown, or pigment, induration of the lungs.

*Etiology.*—Any long-continued mechanical obstruction to the escape of the blood from the lungs into the left cavities of the heart can produce this form of pneumonia. The most frequent and effectual obstruction is furnished by a stenosis of the mitral valves; but any valvular lesion of the heart, dilatation of the ventricles, or aneurism of the arch of the aorta may act in the same way.

*Morbid Anatomy.*—The first effect of the obstruction to the circulation seems to be a change in the capillary vessels in the walls of the air-spaces. These vessels become dilated, tortuous, and have their walls thickened. Then there is a gradual thickening and pigmentation of the walls of the air-spaces; an increase in the size and number of the epithelial cells, until they partly or completely fill the cavities of the air-spaces; an escape of the red blood-cells into the air-spaces. Finally, when the inflammation has reached its full development, there is a smooth,

red hepatization of portions of both lungs, an hepatization due principally to the filling of the air-spaces with epithelial cells.

The lungs are diminished in size, sometimes covered with old adhesions, but seldom with fibrin. There may be more or less serum of dropsical character in the pleural cavities. The texture of the lungs is leathery and dense, or that of a smooth hepatization. They are dry, of a salmon-pink color mottled with brown or black. There may be large or small areas where the air-spaces are filled with extravasated blood.

*Symptoms.*—The principal symptoms exhibited by the patients are those due to the lesions of the heart or aorta. The changes in the lungs do not give as marked symptoms as might be expected. The physical signs are obscure—more or less dulness and diminished breathing. The rational symptoms are—dyspnoea, cough, mucous and bloody sputa, the continued expectoration of pure blood.

*Treatment.*—It is evident that this condition of the lungs is one which cannot be influenced by treatment. We simply attend as well as we can to the disturbances of circulation which have caused the lung trouble.

#### INTERSTITIAL PNEUMONIA.

*Definition.*—A chronic productive inflammation, which involves the connective-tissue framework of the lung and the walls of the air-spaces, and results in the formation of new connective tissue and obliteration of the air-spaces.

*Etiology.*—The well-marked examples of interstitial pneumonia follow acute lobar pneumonia with the production of new connective tissue; broncho-pneumonia; chronic pleurisy with adhesions; chronic bronchitis, and the inhalation of the dust of coal, or of stone.

*Morbid Anatomy.*—The condition of the lungs varies with the cause of the interstitial pneumonia.

(1.) If it follows acute lobar pneumonia with the production of new connective tissue, one lobe, or an entire lung, is covered with pleuritic adhesions, it is small, smooth, and dense; the air-spaces and small bronchi are obliterated by the new connective tissue.

(2) If it follows broncho-pneumonia, one or more lobes are studded with fibrous nodules, or are converted into dense fibrous



tissue. The pleura is thickened, the bronchi are inflamed and often dilated.

(3) If it follows thickening of the pleura, bands of connective tissue extend from the pleura into the lung, the bronchi are inflamed and often dilated.

(4) If it follows chronic bronchitis there are fibrous nodules around the bronchi, with more or less diffuse connective tissue.

(5) If it is due to the inhalation of the dust of coal or stone, we find in both lungs fibrous peribronchitic nodules and diffuse connective tissue.

In most of the cases the portions of lung exempt from the interstitial pneumonia are emphysematous.

*Symptoms.*—The patients have a cough with mucous expectoration. The cough becomes more constant and troublesome as the disease progresses. The expectoration becomes muco-purulent, sometimes fetid. If the bronchi become dilated, the mucus accumulates in them and is coughed up at intervals in large quantities. There may be occasional hæmoptyses. There is dyspnœa on exertion, at first slight, later more marked. There may be uncomfortable feelings or actual pain over the affected side of the chest. There is gradual loss of flesh and of strength. Neither laryngitis nor diarrhœa belong to the disease. There is no fever except with exacerbations of the bronchitis.

The affected side of the chest is retracted, the other side is enlarged, the heart is displaced, the vertebral column is curved laterally. There is, on percussion, dulness, or flatness, or tympanitic resonance. The vocal fremitus is increased. The breathing is sonorous, sibilant, bronchial, cavernous, or amphoric, according to the condition of the bronchi and the degree of consolidation of the lung. There are pleuritic creaking sounds, subcrepitant, coarse, or gurgling râles.

The disease is one which lasts for many years, and the patients usually die with some acute inflammation of the other healthy lung.

*Treatment.*—The most efficient treatment is that the patient should reside permanently in a climate where he is able to live out of doors, and where his bronchitis does well.

If this cannot be done, we attend to the bronchitis and the nutrition of the patient as well as we can.

## TUBERCULAR PNEUMONIA.

*Definition.*—Under this name we include all the inflammations of the lung which are accompanied with the growth of tubercle bacilli. Of such inflammations there are a number which differ from each other widely in their morbid anatomy and clinical symptoms. We are not at the present time provided with satisfactory names to designate these different forms of tubercular pneumonia, so that we still have to use the old arbitrary terms to which there are so many objections.

We distinguish, therefore :

Acute miliary tuberculosis of the lungs.

Chronic miliary tuberculosis of the lungs.

Acute pulmonary phthisis.

Chronic pulmonary phthisis.

## ACUTE MILIARY TUBERCULOSIS OF THE LUNGS.

*Definition.*—A tubercular inflammation of the lungs characterized by the presence of a number of small foci of inflammation, of which the inflammatory products form very small nodules, called miliary tubercles. The tubercular inflammation may be confined to the lungs, or it may be part of a general tuberculosis.

*Etiology.*—For the development of tubercular inflammation in any part of the body there are necessary : the proper predisposition of the individual, a local cause of inflammation, and the growth of tubercle bacilli. In the lungs the tubercle bacilli seem to be either inhaled or brought to the lungs by the blood. It is possible that the bacilli may be first inhaled, then collected in the bronchial glands, and from the glands find their way into the pulmonary circulation. Certainly, we see cases in which tubercular inflammation of the bronchial glands precedes miliary tuberculosis of the lungs. The bacilli which are inhaled must be derived to some extent from the dried sputa of persons who have tubercular pneumonia. The bacilli are much more abundant in the air of some localities than of others, and at high altitudes and on the ocean the air contains none of these organisms. The bacilli which are conveyed to the lungs by the blood are derived from a focus of tubercular inflammation in some other parts of the body. In man tuberculosis is conveyed by inocula-

tion from one person to another only in rare cases. In some animals tuberculosis can be inoculated at will, and miliary tubercles of the lungs easily produced. Trudeau, however, has shown that the success of such an inoculation can be influenced by environment, and that it is possible to keep rabbits under such conditions of good health that they cease to offer a good soil for the growth of tubercle bacilli. The most perfect miliary tubercles in the lungs of animals which I have ever seen, are those produced by injections of dead tubercle bacilli into the trachea of rabbits (Prudden).

*Morbid Anatomy.*—The miliary tubercles may be confined to part of a lung, or be distributed through both lungs. They are close together, or separated by considerable intervals, or aggregated together into larger masses. They are of gray, white, or yellow color. They all contain tubercle bacilli, but these bodies are much more numerous in some tubercles than they are in others. They are composed of :

1. Groups of air-passages and air-vesicles filled with granular matter, pus-cells, and epithelium.

2. Groups of air-passages and air-vesicles of which the walls are infiltrated with tubercle tissue, while their cavities are filled either with tubercle tissue or with epithelium, pus, and fibrin.

3. Infiltrations of the walls of small bronchi with tubercle tissue, or round-celled tissue, the infiltration extending to the walls of the adjacent air-spaces.

4. Nodules of tubercle tissue situated in the pulmonary pleura, the septa between the lobules, the walls of the bronchi, and the walls of the veins.

In addition to the presence of the miliary tubercles the bronchi are congested and coated with mucus, the walls of the air-spaces are congested, the epithelial cells which line them are increased in size and number, some of the air-spaces are filled with epithelium, fibrin, and pus; there may be fibrin on the pulmonary pleura.

*Symptoms.*—1. The method of infection may be such that an enormous number of miliary tubercles are at once formed in the lungs and in other parts of the body, the poisonous effects of the chemical substances produced by the growth of the tubercle bacilli are very marked, and the patients behave as if they had a general disease rather than an inflammation of the lungs.

Although the lungs are congested and thickly studded with

miliary tubercles the physical signs are not as constant or as plain as one would wish them to be. The changes in the percussion-note are not well marked. There may or may not be some dulness. Crepitant or subcrepitant or coarse râles, bronchial voice and breathing, or rude breathing are sometimes present, sometimes absent. Moreover the physical signs may be modified by the existence of old tubercular lesions in the lungs. The rational pulmonary symptoms also are not constant. Cough and mucous sputa, rapid and oppressed breathing, and pain over the chest are present in some cases, absent in others.

The temperature rises rapidly and is between  $100^{\circ}$  and  $107^{\circ}$  F. throughout the disease. The pulse and heart action become more rapid and feeble as the disease progresses. The tongue is coated and soon becomes brown and dry. There is loss of appetite, nausea, and sometimes vomiting. The bowels remain regular, or are constipated, or loose. Sooner or later alternating stupor and delirium, extreme emaciation and the typhoid state are developed. The disease may last for only a few days, or for three or four weeks. So far as I know it always terminates fatally.

2. The method of infection is such that miliary tubercles are formed only in part of a lobe, or are distributed through an entire lobe, or the whole of one lung, or a large part of both lungs. But there are no tubercles outside of the lungs, it is a localized tubercular inflammation.

*Etiology.*—This condition may be developed at any time in a person who has the tubercular disposition, and we are seldom certain in such cases as to the exciting cause of the inflammation or the exact method of the infection. It is also of frequent occurrence in persons who already have a chronic tubercular inflammation of some part of the lung. In such cases it is probable that the infection comes from the old tubercular lesion.

*Morbid Anatomy.*—As the disease runs rather a subacute than an acute course the tubercles are firmer and more regularly composed of tubercle tissue than is the case in acute general tuberculosis. The most frequent position of the tubercles is at the apex of one lung, but they may be localized at any part of the lungs. It is not often that the whole of both lungs is involved. The cases vary greatly as to the extent of the associated bronchitis, pleurisy, and exudative pneumonia. The bronchitis may be confined to the portion of the lung which contains the miliary tuber-

cles, or it may be a general bronchitis involving the larger tubes in both lungs. The pleurisy is either with fibrin alone, or with large quantities of serum in the pleural cavity. The exudative pneumonia may involve only small portions of the lung, or an entire lobe may be consolidated.

*Symptoms.*—These are in proportion to the extent of lung involved. If only a part of one lung is involved the other symptoms are often preceded by one or more hæmoptyses. When the disease is established the patients suffer from :

1. A febrile movement which is regularly higher in the afternoon and followed by sweating at night. The fever may be confined to the afternoon, with normal morning temperatures. The height of the temperature varies with the extent of lung involved, the severity of the associated bronchitis, pleurisy, or pneumonia, the character of the patient, and, probably, the quantity of poisonous products which are evolved by the growth of the bacteria.

2. An increased frequency of the heart action. This is a very constant symptom, so constant that whenever we find a patient with a rapid pulse for which there is no evident cause we must always think of the possibility of a tubercular inflammation of the lungs.

3. The breathing is often increased in frequency and sometimes labored even with lesions of small extent. It is made worse by an extension of the tuberculosis, or by the development of bronchitis, pleurisy, or pneumonia.

4. Most of the patients have more or less cough. They may only cough in the morning, or throughout the day, or in severe attacks, which may provoke vomiting. In some patients the cough is hardly noticeable, in others it is the most distressing feature of the disease. The immediate cause of the cough is not always the same. It may be principally due to a catarrhal pharyngitis, or to the bronchitis, or to the pleurisy. How far the presence of the tubercles in the lungs causes cough is difficult to say. In some persons the cough is evidently largely hysterical, out of all proportion to any real reason for it. The expectoration, if there is any, is mucous, or muco-purulent in character. It often, but not always, contains tubercle bacilli. There may be small hæmoptyses throughout the course of the disease.

5. Loss of appetite with more or less nausea. This is present in a great many of the patients. It is a serious matter, for it is

one reason for their loss of flesh and strength. Actual vomiting usually occurs only after fits of coughing.

6. Loss of flesh and strength are characteristic symptoms, but they are not always in proportion to the extent of lung involved. We must judge of the real condition of the patient rather by the pulmonary lesions than by the general condition.

It is questionable whether the miliary tubercles alone give any physical signs. And as a matter of fact many patients who have acute miliary tuberculosis give no physical signs at all. But the associated bronchitis, pleurisy, and pneumonia do give physical signs. The bronchitis gives coarse and subcrepitant râles; the pleurisy, dulness, friction sounds, crepitant and subcrepitant râles; the pneumonia, dulness, increased vocal resonance, bronchial voice, and bronchial breathing.

(1) When the disease is once established it may continue for a number of months, the inflammation then subsides, and the patients recover altogether.

(2) Or, the inflammation may remain localized but will become chronic, and the patients go on with the history of chronic miliary tuberculosis.

(3) Instead of this the tubercular inflammation, after remaining for months circumscribed, will either continuously, or at intervals, extend and involve more and more of the lungs. When this is the case the patients get worse either continuously, or with intervals of improvement.

(4) In some cases there are one or several intercurrent attacks of bronchitis, pleurisy, or pneumonia so severe as to modify the course of the disease. With each attack of this kind the temperature runs up, the physical signs change, and the patient is evidently more seriously ill.

(5) If both lungs are at the first involved by the tubercular inflammation, the patients have the same symptoms but in a more severe form. They lose flesh and strength rapidly, develop alternating delirium and stupor, pass into the typhoid condition, and die within a few weeks.

*Treatment.*—If both lungs are involved in the morbid process treatment is of no avail. But if only a part of one lung is diseased, the patients can be much benefited by proper management—the two essential points in the treatment being that the patients should eat enough food, and live in a proper climate.

As regards the feeding, it is important that the patients should

take fat in some form in addition to the other articles of diet. In some patients a proper climate alone will improve the appetite; in others, it may be necessary to use bitters, alkalies, or mineral acids, to relieve constipation, to wash out the stomach, or to feed through the stomach-tube. Generally speaking, all the prepared and peptonized foods and extracts are to be avoided. The patients do best with milk, cream, meats, vegetables, fruits, and breadstuffs.

As regards climate, I doubt if there is any one place suitable for all the patients. The idiosyncrasy of each person must be considered, and we find that some do best on the sea-shore, some in the interior, some in a warm climate, some in a cold climate, while some do best if they travel from place to place.

#### CHRONIC MILIARY TUBERCULOSIS.

*Definition.*—A chronic tubercular inflammation of the lungs characterized by the formation of miliary tubercles, to which may be added bronchitis, dilatation of the bronchi, pleurisy, interstitial pneumonia, and emphysema.

*Etiology.*—It seems to be necessary to suppose in these cases: a predisposition, an exciting cause for inflammation, and the growth of tubercle bacilli. It is evident, however, that the mode and character of the infection must differ from those of acute miliary tuberculosis. The structure of the tubercles is different, they contain very few tubercle bacilli, and the other morbid changes in the lungs may be of more importance than are the tubercles.

*Morbid Anatomy.*—The morbid process begins regularly at the apex of one of the lungs, and then slowly extends either progressively or in attacks, until a larger portion of the lungs is involved.

In the simplest form of the disease the only change in the lungs is the formation of miliary tubercles. These tubercles are harder and denser than are those which are found with acute tuberculosis. They are composed of tubercle tissue, or round-celled tissue, or connective tissue, or are in the condition of cheesy degeneration. They contain but few tubercle bacilli.

Another simple form of the disease is when miliary tubercles alone are formed in lungs which are already the seat of vesicular emphysema.

Usually, however, in addition to the miliary tubercles there are other changes in the lungs. These additional lesions begin in the same part of the lung where the tubercles are formed, and accompany the development of the tubercles in fresh parts of the lungs.

(1) There may be a localized catarrhal bronchitis.

(2) There may be an inflammation of the walls of the bronchi with partial destruction of these walls, and the formation of cylindrical or sacculated bronchiectasiæ. The walls of the cavities thus formed may be converted into connective tissue, or they may remain suppurating and necrotic.

(3) There may be an interstitial pneumonia with the production of new connective tissue, the obliteration of the air-spaces, and the consolidation of portions of the lung.

(4) There may be dilatation of the air-spaces of the portions of the lungs which are not consolidated.

(5) There may be thickening of the pulmonary and costal pleura, with connective-tissue adhesions.

(6) While the morbid process begins as a localized tubercular inflammation of the lung, and often retains throughout this local character, yet it may also happen that from this local lesion other parts of the body may be infected. Tubercular laryngitis, and tubercular inflammation of the solitary and agminated glands of the small intestine, often complicate the pulmonary lesion, and sometimes even acute general tuberculosis is produced.

*Symptoms.*—The disease may follow an acute pulmonary tuberculosis, or it may be chronic from the outset. There is such a very great difference in the behavior of the disease in different persons, that it is necessary to arrange the cases into a number of groups according to the character of the lesions and the symptoms.

1. There seems good reason to believe that a small tubercular inflammation of one apex often runs its course and terminates in recovery, without ever giving symptoms of sufficient severity to attract the attention of the patient, or lead him to consult a physician.

2. There are patients who suffer for some time from pulmonary emphysema with its attendant symptoms. In them miliary tubercles are formed in the lungs and slowly increase in number, but are not accompanied with the growth of much connective tissue, and are scattered at some distance from each other through



the lungs. The formation of the tubercles does not change the physical signs belonging to the emphysema which the patient already has. The ordinary rational symptoms of emphysema continue unchanged, but the patients lose flesh and strength rapidly, and finally die very much emaciated. It is very difficult to distinguish these cases from the bad cases of emphysema without tuberculosis.

3. A very common form of the disease is that in which the inflammation is confined to one or both apices, continues for some time, subsides, and the patient recovers.

In these cases the first symptom may be one or more small or large hæmoptyses. Before the bleeding the patients have had no pulmonary symptoms, but after the bleeding these symptoms are gradually developed.

Or the first symptom may be a troublesome cough with little or no expectoration. This cough at first does not seem of much consequence, but it continues in spite of all remedies.

Or the patients simply lose flesh and strength without any evident reason for this loss of nutrition. The pulse is also increased in frequency, but for a time there are no pulmonary symptoms.

At this early stage of the disease there may be no physical signs.

As the disease goes on the patients have more or less cough, which troubles them only in the morning, or occurs in attacks, or is persistent and troublesome through the whole day. There may be no expectoration. When present the sputa are mucous or muco-purulent, not abundant, and often contain tubercle bacilli. There is some dyspnœa on exertion. Some of the patients complain of a great deal of pain over the inflamed lung, others have no pain at all. Hæmoptyses may be repeated at any time, and are apt to be followed by an increase in the severity of the symptoms. The frequency of the pulse is increased in nearly all the patients. A febrile movement is not a prominent symptom, there may be afternoon temperatures of 100° F. followed by sweating, but often during much of the time there is no fever at all. The appetite is poor, nausea and vomiting are often troublesome. The patients lose flesh and strength. In some cases the symptoms are not at any time severe; in others the constitutional disturbances are so out of proportion to the lesion as to indicate systemic infection.

The physical signs become more marked with the further development of the thickening of the pleura, the formation of miliary tubercles, the growth of interstitial connective tissue, the localized bronchitis, and the dilatation of the bronchi. So we find: retraction of the chest-wall above and below the clavicle, the percussion sound of higher pitch and of shorter duration above and below the clavicle, subcrepitant râles, friction sounds, a higher pitched and louder voice, the breathing diminished, or of altered quality, or with prolonged expiration.

Such a circumscribed tubercular inflammation usually continues for a year, sometimes longer, then it subsides and the patients recover. The portion of lung which has been inflamed is left permanently changed into connective tissue.

As the patient has had one attack of tubercular inflammation, so, although he has entirely recovered from this attack, he may have subsequent attacks of the same kind. In some of the patients, after the subsidence of the inflammation, tubercle bacilli are left in the portion of lung which has been inflamed. These may at any time later serve as a source of infection for a new local or general tuberculosis.

4. The tubercular inflammation, beginning at the apex of one lung, gradually extends and involves a large part of both lungs. The pleuritic adhesions become more extensive, a larger number of bronchi are involved in the catarrhal bronchitis, miliary tubercles and new connective tissue replace more and more of the lung tissue. The changes in the walls of the bronchi and of the lung surrounding them result in the formation of bronchiectatic cavities, which constantly increase in size, and the walls of which are necrotic or suppurating. As the disease progresses, therefore, the patients suffer, not only from the infection due to the tubercular inflammation, but also from that due to the necrosis and suppuration of the walls of cavities, while in addition more and more of the lungs is rendered unfit for breathing.

The physical signs of consolidation and of pleuritic adhesions become more marked, and as the cavities are formed and increase in size their physical signs are added.

The cough, which depends at first upon the bronchitis or the pleurisy, is made worse by the formation of bronchiectasiæ. The expectoration becomes more profuse, more purulent and contains portions of dead lung. Bacilli are present, in the sputum in larger numbers. The difficulty in breathing becomes

more troublesome. The patients differ very much as to the presence or absence of pain in the chest. Either large or small hæmoptyses may be repeated at any time. At first the blood comes from the mucous membrane of the bronchi, but after cavities have been formed there may be bleeding from eroded vessels in their walls. This bleeding is apt to be profuse, continuous, and often fatal. The pulse continues to be rapid through the greater part of the disease. The fever becomes higher and more continuous as the disease progresses, especially after the formation of cavities. The appetite is poor and nausea and vomiting are often troublesome. In women menstruation becomes irregular or ceases altogether. The patients get worse from year to year, but often with periods of improvement, and the whole duration of the disease is apt to be very considerable.

After a time in many of the patients a tubercular inflammation of the larynx or of the agminated glands of the small intestines is added and then the loss of flesh and strength are much more rapid.

5. There are cases in which the tubercular inflammation gradually extends until the whole of both lungs are thickly studded with miliary tubercles and in addition there are extensive pleuritic adhesions, but there is little diffuse fibrous tissue, little or no bronchitis, and no dilatation of the bronchi.

The clinical history of these patients is very misleading. They have no cough, no hæmoptysis, no pulmonary symptoms, no fever. There are no physical signs except those belonging to the pleuritic adhesions. But very often the functions of the stomach and intestine are much disordered. The most striking symptom of these patients is their emaciation. This goes on steadily until the patients are mere skeletons, looking as if they were starving to death with a cancer of the stomach. And yet they may really be taking and retaining a considerable quantity of food. But in spite of the food they continue to lose flesh as if they had a malignant disease. The diagnosis of these cases is often extremely difficult.

6. There are cases in which a tubercular laryngitis is responsible for most of the symptoms, the lesions in the lungs being inconsiderable. The upper part of the larynx and the epiglottis are the portions usually involved. There is first a formation of tubercle granula here and there in the stroma of the mucous membrane with more or less catarrhal inflammation. After a

time the tubercle granula and the mucous membrane over them become necrotic, soften, slough, and form ulcers. These ulcers do not heal, but rather increase in size, their floors and walls being formed partly of tubercle, partly of round-celled tissue. The mucous membrane left between the ulcers is thickened and the seat of chronic catarrhal inflammation.

With such a tubercular laryngitis the patients have a cough with muco-purulent expectoration, a changed voice, and sometimes laryngeal dyspnœa. But the thing which troubles them the most is the pain in the throat, which is made worse by swallowing. This may interfere seriously with the feeding of the patient.

The patients lose flesh and strength, but are not confined to bed. Indeed, many years may pass before the pulmonary tuberculosis gives much trouble.

7. In a few cases a very small miliary tuberculosis of the lung is complicated with an extensive tubercular inflammation of the small intestine.

These patients have a little cough, and perhaps the physical signs of a small consolidation at one apex. It is important to remember that they do not necessarily have diarrhœa. But they lose flesh and strength with a rapidity which the pulmonary conditions do not account for.

*Prognosis.*—In the early periods of the disease, with little evidence of systemic infection, we may often expect the complete recovery of the patient. As more of the lungs is involved, as cavities are formed, as the complicating laryngitis and enteritis are developed and the evidences of systemic infection become manifest, the prognosis is worse and worse.

*Treatment.*—The curative treatment of chronic miliary tuberculosis is embraced in two principal things—climate and feeding.

The selection of a proper climate is to be made with reference to the individual rather than to the condition of the lungs. It should be a climate where he feels well, eats well, sleeps well, and gains flesh and strength. If no one climate answers this purpose, the patient should travel from place to place. The climatic treatment should be continued, if possible, for two full years, and for some persons it is necessary that they should pass the rest of their lives in a favorable climate.

The feeding consists in enabling the patient to eat and digest considerable quantities of wholesome food and of fats. To do this

the most minute attention is necessary to the functions of the stomach, the liver, and the intestines. Great care should be taken to avoid the use of all medicines which interfere with the patient's ability to eat and digest food. Wines and spirits will with some persons increase the appetite and the nutrition, with other persons they interfere with digestion and do harm. Often we are much helped by the use of the stomach-tube. Not only can we in this way cure a complicating chronic gastritis, but we can introduce into the stomach much larger quantities of fluid food than the patients are willing to swallow.

It may also be necessary to alleviate symptoms. The cough is not only annoying, but it often interferes with eating and sleeping. It is, therefore, important in each case to determine the principal cause of the cough.

It may be due to a catarrhal inflammation of the nose and throat, or to either a catarrhal or tubercular inflammation of the larynx. These conditions are best treated by local applications made with the spray or with the brush.

It may be due to the pleuritic adhesions. If this is the case counter-irritation by blisters or iodine may be of service, but some of these pleuritic coughs can only be controlled by opium.

For the cough which is due to the bronchitis and to the cavities a great many remedies have been employed. In selecting from these remedies, it is well to prefer those which do not disorder the stomach. Creosote seems to be capable of exerting a real effect on the bronchitis, but, unfortunately, it is apt to disorder the stomach. I prefer to use it by inhalation, or by enema. For inhalation a mixture is made of equal parts of creosote, chloroform, and alcohol. The sponge of a Robinson's inhaler is moistened with a few drops of this mixture, and the patient wears the inhaler all the time except when sleeping or eating. An enema is easily made up of five to twenty drops of creosote with some white of egg and a little water, and this can be used once a day. The different preparations of tar and of turpentine, terebene and terpine hydrate, seem to be of service in some cases. The methodical inhalation of compressed air is highly thought of by some physicians. All sorts of combinations of opium, ipecac, squills, sanguinaria, hydrocyanic acid, chloroform, senega, etc., are given as cough mixtures. The mineral acids, nux vomica or strychnia and potassium iodide may somewhat control the bronchitis.

If the patients are anæmic they may be benefited by one of the preparations of iron.

The hemorrhages from the bronchi may be small or large, but even if large and continued for several days they are very seldom fatal. They do, however, weaken the patient very much, and are often followed by an extension of the tubercular pneumonia. To check such hemorrhages it is customary to use hypodermic injections of morphine or of ergotine, or to give by the mouth five grains of gallic acid every two hours, twenty drops of fluid extract of hydrastis every three hours, or one grain of ipecac every hour.

The hemorrhages which come from eroded vessels in the walls of cavities cannot be controlled.

The fever and night-sweats may be made less severe by the use of antifebrin or phenacetine alone, or combined with arsenic or quinine; by the mineral acids, belladonna, or the oxide of zinc; by sponging off the body with hot water at night.

For the diarrhœa we employ a restricted diet, and a number of drugs. The drugs most frequently employed are: the preparations of mercury, ipecac, iron, arsenic, camphor, acetate of lead, bismuth, castor-oil, opium, and naphthalin. These drugs are used singly or combined in different ways.

#### ACUTE TUBERCULAR PHTHISIS.

*Synonyms.*—Acute catarrhal phthisis. Acute consumption.

*Definition.*—An acute affection of the lungs characterized by the association of tubercular inflammation with other forms of inflammation—either exudative or productive, or both. The name is an arbitrary one, and is used for convenience to group together a set of clinical cases.

*Etiology.*—An attack of acute phthisis may follow some previous tubercular inflammation of the lung, or it may be a primary inflammation. A person who has the tubercular predisposition, when exposed to the ordinary causes of inflammation of the lung, and at the same time infected with tubercle bacilli, instead of having a simple exudative or productive pneumonia, has an inflammation of the lung, partly tubercular, partly exudative, partly productive.

*Morbid Anatomy.*—The inflammation of the lungs may follow one of several types, all of which have much the same clinical history, but vary in their physical signs.

1. One or more lobes are completely consolidated. The consolidation is effected by the filling of the air-spaces and small bronchi with epithelium, fibrin, and pus. Scattered through the consolidation are miliary tubercles. The pleura is coated with fibrin.

2. There is a general catarrhal bronchitis and a tubercular inflammation of the walls of some of the bronchi and of small zones of air-spaces immediately surrounding them. The lung is not consolidated, but a section of it appears to be studded with little nodules, each nodule is the section of a bronchus with thickened wall and surrounded by a zone of filled air-spaces.

3. There is a general catarrhal bronchitis, a tubercular inflammation of the walls of some of the bronchi and of the air-spaces which surround them, but in addition there are small or large areas of diffuse consolidation due to the filling of air-spaces with epithelium, pus, and fibrin. The pulmonary pleura is often coated with fibrin.

4. Besides the tubercular broncho-pneumonia, the diffuse consolidation, and the pleurisy, small or large portions of the inflamed lung die. These dead portions of lung first pass into the condition of coagulation necrosis, and then undergo cheesy degeneration. They may remain in the condition of cheesy degeneration for a long time and become surrounded by zones of tubercle tissue or of round-celled tissue; or they often break down and form ragged cavities which communicate with the bronchi.

5. In addition to the lesions already mentioned, the walls of the bronchi are so changed by the tubercular inflammation that cylindrical or sacculated bronchiectasiæ are formed.

*Symptoms.*—The invasion may be acute or subacute.

1. The acute cases. The patients are suddenly attacked with chills, fever, pain in the side, cough with mucous expectoration, and marked prostration. The appearance of the patient is like that of a person attacked with lobar or broncho-pneumonia, and we are often in doubt at first as to the true nature of the disease. One symptom of the invasion, however, is not often seen except with phthisis and that is the bleeding from the bronchi. For a day, or for several days, many of the patients cough up very considerable quantities of blood. Within a few days we begin to get the physical signs, which will be found to vary according to the anatomical condition of the lung. If there is complete consolidation of one or more lobes with fibrin on the pleura we get dul-

ness on percussion, bronchial voice and breathing, and subcrepitant and crepitant râles. If there is only broncho-pneumonia without consolidation, we get sibilant and sonorous breathing and coarse and subcrepitant râles. If there is broncho-pneumonia with areas of diffuse consolidation, we get sibilant and sonorous breathing, coarse and subcrepitant râles, and small areas over which there are dulness on percussion, increased voice, and a crepitant râle are found. The patients continue seriously ill and with high temperatures for one or two weeks. Then there is a partial subsidence of the symptoms. After this the patients may :

(a) Continue to get worse. The fever continues, the cough is very troublesome and accompanied with muco-purulent expectoration, there is rapid loss of flesh and strength, the patients pass into the typhoid state with alternating delirium and stupor. The physical signs which existed earlier in the disease continue and there are added the coarse and gurgling râles which accompany the dilatation of the bronchi and the softening of the dead areas of consolidation. These patients die at the end of a few weeks or months.

(b) Improve very considerably. The temperature falls, the expectoration diminishes, the cough is less frequent, the appetite returns. The patients gain flesh and strength, they are able to leave the bed, and later the house, but yet they are not well. A considerable portion of the lung remains diseased and the patients go on to the condition of chronic phthisis.

(c) Recover. Of the lesions of acute phthisis, the tubercular changes, the death of portions of lung, and the dilatation of the bronchi are necessarily permanent. On the other hand, the catarrhal bronchitis may subside, the epithelium, pus, and fibrin within the air-spaces may degenerate and be absorbed. It is possible, therefore, for the patients who have only catarrhal bronchitis, exudative pneumonia, and comparatively little tubercular pneumonia to recover. We see this in two sets of cases. First, the patients who have consolidation of an entire lobe due to the filling of the air-spaces with epithelium, pus, and fibrin, and to the presence of miliary tubercles. The epithelium, pus, and fibrin can be absorbed, the tubercles are converted into fibrous tissue, and the patients get well with a lung which is normal except for the presence of fibrous nodules. Second, the patients who have tubercular broncho-pneumonia without de-



struction or dilatation of the bronchi, and without areas of diffuse consolidation. The patients can get well with lungs which are normal except for the presence of a number of peri-bronchitic fibrous nodules.

2. The subacute cases. The extent of lung at first involved is small, but gradually increases in size. The patients usually have cough with mucous or muco-purulent sputa which contain bacilli, but sometimes there is very little either of cough or of expectoration, and that even while cavities are being formed. The difficulty in breathing increases with the extent of lung involved, pain over the chest is present in some cases, absent in others.

There is regularly a rise of temperature in the afternoon with sweating at night, and the temperature is higher and more continuous after cavities with suppurating walls are formed. But we have to become accustomed to very great discrepancies between the lung lesion and the height of the temperature, and even to find no fever with an advancing consolidation of the lung.

There may be bleeding from the bronchi or from eroded vessels in the walls of cavities. A large hemorrhage from the bronchi is apt to precede the inflammation of a fresh portion of the lung. The patients, as a rule, have no appetite and gradually lose flesh and strength. But in some persons the changes in the lung for some time produce very little effect on the general health. And it is sometimes very curious, and of importance in judging of treatment, to see patients eating well and gaining flesh with consolidation of an entire lung and without any real improvement in their tubercular pneumonia.

The physical signs are those of the pleurisy, the consolidation, the bronchitis, and the cavities.

The patients are for the most part not sick in bed, and the disease progresses either continuously or in attacks.

Some of the patients go on to have chronic phthisis. Some of them recover, but with lungs more or less permanently damaged. Often these patients can only escape fresh attacks by remaining permanently in a favorable climate.

*The Prognosis* of acute phthisis is unfavorable. Complete recoveries are rare, but the number of patients who go on living with damaged lungs for many years is considerable.

*Treatment.*—While the inflammation of the lungs is active the

patients are to be kept in bed, on a fluid diet, the hæmoptysis controlled by ergot, hydrastin or ipecac, the patients made more comfortable by opium or the bromides.

As the acuteness of the inflammation subsides, the patients return to solid food, they get out of bed, and the question of a suitable climate for them has to be determined.

In some cases it is evident that the changes in the lungs are so extensive and profound that no improvement can be expected; these patients are best kept at home.

In some cases the extent of lung involved is comparatively small, so that we may hope for complete or incomplete recovery. The proper climate for these patients is a dry, inland one, where they are not likely to have fresh attacks of inflammation of the lung. Whether this climate should be a warm or a cold one, must be determined by the character of the individual. In the cases of tubercular broncho-pneumonia without consolidation a cold, dry, inland climate seems to be the best.

The symptomatic treatment is the same as that used in the cases of acute miliary tuberculosis.

#### CHRONIC TUBERCULAR PHTHISIS.

*Definition.*—A chronic affection of the lungs characterized by tubercular inflammation associated with productive and exudative inflammation.

*Etiology.*—Chronic phthisis regularly succeeds acute or sub-acute phthisis, but it may also follow acute or chronic miliary tuberculosis of the lungs, or a tubercular inflammation of some other part of the body.

*Morbid Anatomy.*—The changes in the lungs are of the same nature as those found in acute phthisis, but modified by the long duration of the inflammation. The pleura is coated with successive layers of fibrin, or thickened by the growth of new connective tissue, or covered with adhesions. More or less of the lung is consolidated. This consolidation is effected partly by the filling of the cavities of the air-spaces with inflammatory products, partly by a growth of new tissue in the walls of the air-spaces and between them.

When the hepatization is effected only by changes within the cavities of the air-spaces, the affected portions of lung are solid, increased in size of red, gray, white, or yellow color. When the

hepatization is due to interstitial inflammation the affected portion of lung is dense, but may still be partly aerated ; it is diminished in size and looks like fibrous tissue or granulation tissue, it is often changed in color by the deposition of black pigment. The combination of intra-alveolar and interstitial pneumonia with dilatation of the bronchi and the formation of cavities gives a great variety of pictures.

We find some of the air-spaces filled with large epithelial cells either well formed or fatty ; some with an amorphous granular matter, or a peculiar translucent coagulated substance ; some with fibrin, pus, and epithelium either fresh or in the condition of cheesy degeneration ; some with new connective tissue. The walls of these air-spaces remain unchanged, or they are compressed and the blood-vessels obliterated, or they are thickened.

The interstitial inflammation affects the walls of the air-spaces, the bronchi, the blood-vessels and the septa between the lobules. It results in the production of new connective tissue, of round-celled tissue and of tubercle tissue, either separately or together. By this growth the air-spaces are compressed, deformed, and obliterated in a variety of ways.

The walls of some of the bronchi are infiltrated with round cells or with tubercle tissue. This infiltration is not symmetrical, but affects a bronchus in some particular portion of its length, and in this portion some parts of the circumference of the bronchus are affected more than others. As a result of this irregular infiltration the wall of the bronchus yields here and there, and small sacculated dilatations are formed. In some cases, especially in chronic miliary tuberculosis, the cavities thus formed simply became larger and larger, compressing the surrounding lung. More frequently, however, the process extends from the wall of the bronchus to the surrounding air-vesicle, so that the bronchus is surrounded with tubercle tissue, round-celled tissue, and air-vesicles filled with inflammatory products. Then necrosis sets in, with softening of the walls of the bronchus and of the surrounding inflamed lung. So cavities are formed partly by destruction of tissue, partly by dilatation of bronchi of which the walls are either necrotic or suppurating.

Some of the cavities in chronic phthisis seem to be formed simply by the softening of areas of coagulation necrosis, but the larger number are bronchiectasiæ such as have just been described.

The cavities which are formed by dilatation without necrosis of their walls may be developed with hardly any cough or expectoration. As we examine the patients from time to time, the change in the percussion and the breathing show the increasing size of a cavity which remains nearly dry and empty.

The cavities formed both by dilatation and necrosis, on the contrary, are regularly accompanied by an harassing cough and profuse expectoration. They contain pus, mucus, fragments of dead tissue, and great numbers of tubercle bacilli. Their walls are ragged and irregular, partly necrotic, partly suppurating. In this active condition the cavities may remain up to the time of the patient's death. Or, instead of this, the active processes may subside, the production of pus and the death of tissue cease, the cavity become dry and its walls changed into fibrous tissue. The natural tendency of all these cavities is to increase in size and open into each other.

Miliary tubercles are scattered through the inflamed lung in varying numbers.

The tubercle bacilli are found principally in the walls of the cavities and in the inflammatory products which have undergone cheesy degeneration.

*Symptoms.*—The cough depends principally upon the bronchitis and the morbid processes going on in the walls of cavities. The expectoration consists of mucus, pus, and fragments of dead lung tissue, with many tubercle bacilli. But in patients with consolidation of the lung without bronchitis, and with cavities of which the walls are comparatively healthy, the cough and expectoration amount to very little. Generally speaking, however, the quantity of the sputa and the number of the bacilli are a fair test of the activity of the morbid process.

Hæmoptysis occurs in a large proportion of the cases, and at any time in the course of the disease. After some of these bleedings the patients feel more comfortable, but after others there is a rapid extension of the disease. Hemorrhages from eroded vessels in the walls of cavities are very dangerous.

In some of the patients the pleurisy from time to time gives pain. In others there is a considerable exudation of serum in one of the pleural cavities, which increases the difficulty in breathing. The softening of a dead portion of lung or the rupture of the wall of a cavity just beneath the pleura may cause perforation, pneumothorax, and then either a pleurisy with effu-

sion, or an empyema. At the time of the perforation the patients feel as if something had given way, and at once suffer from the most urgent dyspnœa. The heart's action becomes rapid and feeble, and the veins throughout the body congested. The characteristic physical signs are soon evident. Such a perforation usually proves fatal within a few days or weeks.

The dyspnœa on exertion increases with the extension of the disease and the consequent diminution in the extent of lung available for breathing.

Tubercular laryngitis occurs later in the disease with chronic phthisis than with chronic miliary tuberculosis. It gives rise to cough, hoarseness, and pain.

The fever and the sweating at night seem to be related to the severity of the bronchitis, and of the necrosis and suppuration of the cavities. The temperature rises and falls according to the activity of these conditions. The consolidation of the lung alone can extend with little or no fever.

The functions of the stomach and liver are sooner or later affected, either with or without chronic catarrhal gastritis and fatty infiltration of the liver. Loss of appetite, repugnance to food, nausea, vomiting, and gastric pain may seriously annoy the patient.

Toward the close of the disease a severe diarrhœa often sets in, and the patients lose flesh and strength very rapidly. In these patients after death we may find tubercular ulcers of the small intestine or only a catarrhal colitis. It may also happen that extensive tubercular ulcers of the small intestine exist without any diarrhœa at all.

Tubercular meningitis, or peritonitis, or nephritis may occur as complicating inflammations.

Chronic degeneration of the kidney, or chronic nephritis with or without exudation, are often developed after the phthisis has lasted for some time.

In women menstruation is either irregular, or stops altogether.

The physical signs with only a small area of lung consolidated are: dulness on percussion, broncho-vesicular or feeble breathing, increased vocal resonance, increased bronchial whisper, and in addition, subcrepitant râles and friction sounds. As more of the lung is consolidated the dulness on percussion becomes more marked, the voice and breathing approach more nearly to the

bronchial character, and an increasing bronchitis gives coarse and subcrepitant râles. After cavities have been formed the percussion sound changes to flatness, tympanitic resonance, or the cracked-pot sound. The breathing and voice remain of bronchial quality, or become cavernous. There may be gurgling râles.

The patients regularly lose flesh and strength in proportion to the extent of lung diseased. But it is not rare to see persons who are well nourished and comparatively strong with extensive changes in the lungs.

If we compare chronic miliary tuberculosis with chronic phthisis, we may say that in the former there are but few tubercle bacilli in the sputa or in the lungs, but little necrosis or suppuration, and less consolidation of the lung, but a loss of health and nutrition much greater than would be expected from the extent of the pulmonary lesion.

In chronic phthisis, on the other hand, the number of tubercle bacilli in the sputa and in the lungs is large, necrosis and suppuration are regularly present, the patients seem to suffer rather from septic than from tubercular infection, the severity of the symptoms is usually in direct relation with the extent of lung inflamed.

*Prognosis.*—It is possible for the inflammatory and necrotic processes which belong to chronic phthisis at any time to cease. When this happens, all symptoms of pulmonary disease may also cease and the patients are apparently cured. The portions of lung, however, which have been destroyed or converted into fibrous tissue are never replaced by lung tissue, so that the injury inflicted on the lungs is a permanent one. The fibrous tissue and cheesy masses left behind after the subsidence of active changes are liable to act as foci, from which fresh attacks of inflammation and fresh infection may proceed. The prognosis is, therefore, unfavorable, although life may be prolonged in comparative comfort for many years.

*Treatment.*—Cases of chronic phthisis are to be managed in the same way as are the cases of chronic miliary tuberculosis.

In a work of this kind it is not necessary to describe the plans of treatment by such agents as sulphuretted hydrogen, tuberculin, chloride of gold, etc. None of them have as yet proved satisfactory.

## SYPHILITIC PNEUMONIA.

In children who have inherited syphilis, a number of inflammations of different parts of the body are liable to be developed soon after birth. The lungs are not exempt. They may be studded with gummy tumors of different sizes, or they may be the seat of interstitial, or of intra-alveolar pneumonia.

The interstitial pneumonia may cause the consolidation of one or more lobes. The section of such a consolidated lobe is smooth and of grayish or white color. The consolidation is effected by a growth of new tissue in the walls of the air-spaces, the bronchi, and the blood-vessels, together with the filling of some of the air-spaces with epithelium.

The intra-alveolar pneumonia also produces a whitish hepaticization of considerable portions of the lung. But the consolidation is effected entirely by the filling of the air-spaces with fatty epithelium.

The clinical symptoms in these children are obscure. They often have other syphilitic lesions besides those in the lungs, and become weaker and more emaciated from day to day, without any pulmonary symptoms except the physical signs of the consolidation.

In adults, syphilitic inflammations of the lung are rare. The best collection of cases that I have seen is that given by Hiller in the *Charité Annalen* for 1884. He gives 58 cases of unmistakable syphilitic pneumonia.

*Morbid Anatomy.*—The inflammation is of productive character with the formation of round-celled tissue, of gummy tumors, and of new connective tissue. There may also be some exudation into the air-spaces and a formation of epithelial cells in the air-spaces. The new tissue is formed in the walls of the bronchi, the walls of the air-spaces, the walls of the blood-vessels, the septa between the lobules and the pleura. The new tissue is of low vitality and may become necrotic. There results from such an inflammation, therefore : stenosis, ulceration or dilatation of the bronchi, consolidation of parts of the lung, obliteration of the blood-vessels, lobulation of the lung, and thickening of the pleura.

The gross appearance of the lungs will accordingly vary in different cases.

1. There is an interstitial inflammation beginning at the root of the lung around the large bronchi and blood-vessels. This causes stenosis of the bronchi, consolidation of the lung, or masses of fibrous tissue along the lines of the bronchi. The cases vary as to whether the stenosis of the bronchi or the consolidation of the lung is the principal feature.

2. The inflammation follows the type of a broncho-pneumonia, with thickening of the walls of the bronchi and small zones of peri-bronchitic pneumonia.

3. There are large or small irregular masses, or bands, of dense fibrous tissue in any part of the lung. These replace the lung tissue, and in them may be cavities formed by the dilatation of the bronchi.

4. With the interstitial pneumonia there may be associated the formation of gummy tumors, or an obliterating endarteritis with areas of necrosis.

5. With more or less interstitial pneumonia at the roots of the lungs there is a syphilitic inflammation of the walls of the large bronchi, the trachea, and the larynx. These walls are thickened in some places, ulcerated in others, so that in some places there is stenosis, in others dilatation.

6. We also occasionally meet with pneumonia of the anatomical type of ordinary lobar or broncho-pneumonia. But the clinical history although acute is irregular, and it is probable, although not at all certain, that they are caused or modified by the syphilitic poison.

*Symptoms.*—Syphilitic pneumonia is one of the later manifestations of syphilis, in most of the cases coming on several years after the initial lesion. A great many of the patients have other syphilitic lesions, a fact of much assistance in making a diagnosis which is always difficult.

Of the pulmonary symptoms perhaps the most constant is dyspnœa. This is like any dyspnœa due to narrowing of the trachea or large bronchi. First a dyspnœa on exertion, then a constant dyspnœa made worse by the least bodily or mental exertion, and becoming more and more distressing up to the time of the patient's death.

Cough is present at some time in most of the cases, a dry cough, a laryngeal cough, or a cough with mucus or muco-purulent expectoration. Small hæmoptyses occur from time to time in some cases.



Pain referred to some part of the chest is present in some cases, absent in others.

A febrile movement seems to be the exception rather than the rule.

If a syphilitic laryngitis exists the symptoms belonging to this will be added to those of the pneumonia.

The physical signs vary with the exact condition of the lungs and are often obscure. They depend upon the pleurisy, the bronchitis, the stenosis or dilatation of the bronchi, and the consolidation of the lung.

So in the different cases we may get tubular breathing over one or both lungs, absence of breathing over one lung or part of a lung, subcrepitant, coarse, or gurgling râles, usually localized, dulness on percussion, and increase in vocal resonance according to the extent of the consolidation.

The fact that the inflammation involves the roots of the lungs rather than their apices causes the physical signs to be heard largely over the central portions of the lungs, while in tubercular inflammations of the lungs it is over the upper lobes that the physical signs are usually heard.

The symptoms continue for weeks or months, the patients gradually lose flesh and strength, and finally die from the interference with breathing or worn out with the disease.

*The Diagnosis* is apt to be difficult. The symptoms resemble those of chronic tuberculosis, of aneurism of the aorta, of intrathoracic tumors, and of actinomycosis of the lung. We are very dependent upon the history of the patient and the presence of other syphilitic lesions.

*Treatment.*—It is natural in these patients to adopt an energetic treatment with mercury and the iodide of potash, although the rule seems to be that the disease is fatal.

## GANGRENE OF THE LUNG.

*Definition.*—Death accompanied by putrefaction of a portion of the lung.

*Etiology.*—Whenever the vitality of a portion of the lung is impaired and at the same time the bacteria of putrefaction are present, there may be gangrene. It is found, therefore, with lobar pneumonia, hæmorrhagic infarctions, compression or embolism of the pulmonary or bronchial vessels, wounds of the lung, con-

tusions of the chest, cavities in the lung, foreign bodies in the bronchi, cancer of the œsophagus, and in persons whose health has been enfeebled by disease or privation. But it also occurs without discoverable cause in persons who have been in good health.

*Morbid Anatomy.*—Gangrene of the lung is either circumscribed or diffuse.

Circumscribed gangrene occurs in the form of one or more foci of small size where the lung is of blackish or greenish color, soft, or broken down into ragged cavities. These foci have a most offensive odor. The lung around them is inflamed and the air-spaces contain epithelium, fibrin, and pus. Such gangrenous foci when once formed may increase in size; as they do so the adjoining veins may become filled with infectious thrombi, or eroded. From the thrombi infectious emboli can be carried into the circulation and set up inflammatory foci in different parts of the body. From the eroded vessels there are considerable hæmorrhages. If the spot of gangrene is near the pleura it may set up either a simple or a gangrenous pleurisy. Or the pulmonary pleura may be perforated and pyopneumothorax result. Intense bronchitis, either catarrhal or croupous, may be excited by the irritation of the gangrenous matter. If the patients recover the gangrenous portion of lung is entirely removed, a cavity is formed of which the walls are changed into connective tissue. Such a cavity may remain for a long time, or it may become contracted.

Diffuse gangrene may be secondary to the circumscribed form, or it may be diffuse from the first. The greater portion of a lobe, a whole lobe, or even a whole lung, may be involved. The portion of lung involved is changed into a soft, foul-smelling, blackish or greenish mass.

*Symptoms.*—The patients have a cough with more or less fetid expectoration and a fetid breath. There is much variety as to the quantity of the expectoration, it may be scanty or abundant. When it is abundant and is allowed to stand for a time in a glass dish it separates into three layers: The upper, frothy, opaque, and of a dirty gray or yellowish color; the middle, clear and watery; the lower, greenish and purulent, or mixed with blood. It consists of serum, mucus, pus, and shreds of lung tissue. There may be, however, only a very fetid breath without any expectoration. If the pulmonary vessels are eroded large quantities of blood are coughed up.

The patients have an irregular fever, they lose flesh and strength and pass into the septic condition. But some of the patients, who apparently have gangrene of the lung and recover, are not at any time so sick as one would expect.

The physical signs are sometimes obscure, sometimes well marked. They are most commonly found over the middle of the chest behind. At this point we may get dulness or flatness on percussion ; absence of breathing, bronchial breathing, or cavernous breathing ; exaggerated or bronchial voice, and coarse râles.

If the pleura is inflamed or perforated, producing pneumothorax, the symptoms belonging to these conditions are added.

The diagnosis is often difficult. It may be evident that the patient has a serious disease and yet impossible for some time to determine its character. Even when it is certain that there is a pulmonary lesion we cannot always tell whether this is gangrene, abscess, or fetid bronchitis.

*The Prognosis* is always serious, but recovery is by no means impossible.

*Treatment.*—Besides the employment of such measures as add to the comfort of the patients and contribute to their nutrition, it is customary to give creosote or carbolic acid either by inhalation or by the mouth. Perhaps the simplest plan is to use a Robinson's inhaler moistened with equal parts of creosote, alcohol, and chloroform.

In a moderate number of cases gangrenous cavities in the lungs have been opened and drained.

## ASTHMA.

*Definition.*—An affection characterized by paroxysmal dyspnoea, recurring at intervals, generally in the night, the dyspnoea due to a contraction of the bronchi.

The same name of asthma is also frequently employed to designate the paroxysmal dyspnoea caused by disease of the heart, and by contraction of the arteries.

*Ætiology.*—Bronchial asthma occurs most frequently in persons who have pulmonary emphysema, but it is by no means rare in persons whose lungs are normal except for the condition of the bronchi.

The causes which produce an attack of asthma may act directly on the mucous membrane of the bronchi, or indirectly

on the bronchi through the blood, or the nervous system. So we find some persons who never have an attack of asthma except when exposed to a directly exciting cause ; while other persons have constantly recurring attacks for long periods of time without any direct cause for each attack.

Among the direct causes we reckon bronchitis, inflammations, and obstructions of the nose, climatic influences, dust, vegetable irritants, chemical vapors, and animal emanations.

The ordinary dust floating in the air, the odor or pollen of many plants and grasses ; the vapors of pitch, sulphur, or phosphorus ; the peculiar smell of dogs, cats, or horses are familiar examples of direct causes.

The effect of climate in causing asthma is very marked in some persons. This effect does not follow any definite law, but only the idiosyncrasy of the individual. It does not matter whether the locality is warm or cold, wet or dry, low or elevated. For nearly every asthmatic person we can find some one place where he will have little or no asthma.

Of all the causes of asthma, however, bronchitis is the most frequent. In the patients who belong to this class the bronchitis constitutes the important part of the case, for if the attacks of bronchitis can be prevented there is no more asthma.

Of late years much attention has been called to diseases of the nasal passages as a cause of asthma. I think there is no question that they do constitute one of the causes. But it is going a great deal too far to say that they are the only cause.

Among the indirect causes of asthma we enumerate mental emotions, indigestion, hysteria, gout, heredity, and some of the skin diseases. But it must be confessed that we are often unable to say why a previously healthy person should at some particular time, without any exciting cause, begin to have attacks of asthma.

*Morbid Anatomy.*—As asthma is a functional disease it has no lesions. But in the bodies of old asthmatics we commonly find the morbid changes belonging to pulmonary emphysema and bronchitis.

*Symptoms.*—A paroxysm of asthma begins with a feeling of oppression or suffocation about the upper part of the chest which obliges the patient to sit up in order to breathe. The feeling of suffocation continues, and the patients bring into play all the muscles of respiration in order to satisfy the hunger for air. The skin becomes livid, the pulse feeble, and the patient's face shows

his suffering. If we listen to the chest, we hear over both lungs the sibilant and sonorous breathing caused by the contraction of the bronchi. Such an attack lasts for hours or days. During the most severe attack the patients look as if they might die at any minute, but yet after a time the attack always subsides.

As attacks of asthma are due to a variety of causes, so they present themselves to us under a variety of clinical aspects.

1. There are the persons who never have asthma unless they have an attack of acute bronchitis. In such persons we have to look at the bronchitis as the real disease, while the asthma is only a complication.

2. There are the persons who only have asthma at certain times of year and in certain localities, the attacks being caused by the inhalation of the pollen or odor of plants. These persons are said to suffer from "hay fever," "rose cold," "autumnal catarrh," etc.

3. There are persons in whom the asthma only constitutes one of the symptoms of pulmonary emphysema. These cases will be described with emphysema.

4. There are other cases in which the asthma occurs by itself as a pure neurosis. In these persons the disease is apt to be very tenacious, the paroxysms recurring again and again even after considerable intervals of improvement. In the more severe cases the bronchi are somewhat contracted and the breathing labored nearly all the time, while the spasmodic dyspnoea recurs at regular intervals. The patients become worn out by the constant dyspnoea, the face is one of suffering, the chest is bent forward and stooping, the nutrition is impaired, the whole condition is one of chronic invalidism, but yet life is prolonged and death is usually due to some other disease.

*Treatment.*—The objects of treatment are: to cut short the attacks of asthma, and to prevent the recurrence of subsequent attacks.

To cut short an attack of spasmodic asthma we employ such means as will relax the spasmodic contraction of the walls of the bronchi. This can be done in a variety of ways: inhalation of the fumes of stramonium, nitrate of potash, chloroform, ether, or the nitrite of amyl; hypodermic injections of morphine, or chloral hydrate given by the mouth or the rectum; the use of the drugs which increase the production of mucus from the bronchi, such as lobelia and *grindelia robusta*.

To prevent the recurrence of the attacks we examine into the condition of the nasal passages to see whether there is disease there which may cause the asthma. We inquire into the history to determine whether the asthma is not caused by bronchitis, or by the pollen or odor of plants.

If the asthma is a pure neurosis a considerable number of patients can find a climate in which they cease to suffer. But the selection of this climate has to be made experimentally by each patient. There is no rule to guide us. Each person has to travel from place to place until they find the particular spot where they cease to have asthma.

For the patients who cannot travel the most efficient treatment seems to be the long-continued administration of the iodide of potash, the systematic inhalation of compressed air, attention to the feeding, disorders of digestion, and to any conditions which impair the general health.

### HÆMOPTYSIS.

Blood which is coughed up comes for the most part from the bronchi; less frequently from the pharynx, from eroded vessels in the walls of cavities, or from aneurisms of the pulmonary artery or of the aorta.

Hæmoptyses occur so frequently with the tubercular inflammations of the lungs and so much less frequently with other morbid conditions, that any hæmoptysis is regarded with a good deal of suspicion.

In tubercular inflammations of the lungs the bleeding is from the mucous membrane of the bronchi. The quantity of blood coughed up may be large or small. The bleeding may last only a few minutes, or continue a number of days. The same patient may have only a single hæmoptysis, or several. There seems to be no period in the course of tubercular inflammation of the lungs which is exempt from the liability to bleeding from the mucous membrane of the bronchi. Especially is it to be remembered that either large or small hæmoptyses may precede by a considerable interval of time any rational symptoms or physical signs of pulmonary disease.

In the older cases of pulmonary tuberculosis in which cavities have been formed, the vessels in the walls of these cavities may be eroded, with an escape of blood which is large and dangerous

The very frequency of the association of hæmoptysis with pulmonary tuberculosis makes it important to enumerate the other conditions under which hæmoptysis may occur. The following are the forms of hæmoptysis which occur without pulmonary tuberculosis:

1. A person has an attack of hæmoptysis only lasting for a short time, but during which he raises a considerable quantity of blood. He has no other attacks and does not develop any pulmonary lesions. The bleeding may follow severe muscular exertion, great mental excitement, or occur without discoverable cause.

2. In women hæmoptysis may take the place of menstruation. Flint says that he has seen hæmoptysis occurring at regular intervals for four years after the suspension of the menses. But these cases must be looked on with suspicion. A woman may cough up blood on several occasions instead of menstruating and then go on to have lung disease.

3. Chronic naso-pharyngeal catarrh is sometimes attended with occasional small hæmoptyses.

4. There are a set of women who are hysterical, anæmic, always suffering from some real or fancied ailment, who from time to time cough up a little blood.

5. It is said that pregnant and nursing women sometimes have hæmoptysis.

6. With disease of the aortic and mitral valves, especially with mitral stenosis, bleeding from the bronchi is of frequent occurrence. In the course of the heart disease the patients from time to time, during periods of several days, cough up clear blood in considerable quantities.

7. Aneurisms of the branches of the pulmonary artery within the lungs, when they rupture, cause fatal hemorrhage, a large part of the blood being coughed up.

Aneurisms of the arch of the aorta which erode the trachea or main bronchi may rupture into the trachea or bronchi by small or large openings. With the small openings the patients cough up a little blood from time to time, while part of the escaped blood is inspired into the lungs and sets up a peculiar form of pneumonia. With the large openings the blood escapes through the trachea in enormous quantities, and the patients bleed to death within a few minutes.

8. Patients who suffer from emphysema and chronic bron-

chitis not infrequently from time to time cough up small quantities of blood. Much less often such patients have at some time a large bleeding from the bronchi. Part of this blood is coughed up at once, part is coagulated in the large bronchi and is afterward coughed up in the form of casts of these tubes.

9. Sir Andrew Clark (*Lancet*, 1889, p. 841) describes a form of bleeding which he calls "arthritic hæmoptysis," of which he says that he has seen some twenty cases. He lays down the following propositions: There occurs in elderly persons, free from ordinary diseases of the heart and lungs, a form of hæmoptysis arising out of minute structural alterations in the terminal blood-vessels of the lung.

These vascular alterations occur in persons of the arthritic diathesis, resemble the vascular alterations found in osteoarthritic articulations, and are themselves of an arthritic nature.

Although sometimes leading to a fatal issue, this variety of hæmoptysis usually subsides without the supervention of any coarse anatomical lesion of either the heart or the lungs.

This variety of hemorrhage is aggravated or maintained by the frequent administration of large doses of strong astringents, and by an unrestricted indulgence in liquids to allay the thirst which the liquids themselves create.

The treatment which appears to be the most successful in this variety of hæmoptysis consists in diet and quiet, in the restricted use of liquids, and the stilling of cough; in calomel and salines, in the use of alkalies, with iodide of potassium, and in frequently renewed counter-irritation.

10. Dr. Flint ("Practice of Medicine," p. 265) says that he has met with a few cases of persistent bronchial hemorrhage. In two of these cases, after expectoration daily more or less of a sero-sanguinolent liquid during several months, recovery took place under the use of tonics and hygienic measures. In the third case the hemorrhagic expectoration continued for six years, during which time repeated examinations of the chest failed to discover any positive signs of pulmonary disease.

11. Severe injuries inflicted upon the wall of the thorax may be followed by the expectoration of blood for hours, or days.

*Treatment.*—In managing cases of bleeding from the mucous membrane of the bronchi it is important to bear in mind that even the most profuse hemorrhages are seldom fatal.

The methods of treatment commonly employed are: the appli-



cation of cold to the chest, the temporary ligation of one of the arms or legs, the internal use of opium, ergot, hydrastis, kramaria, tannic acid, gallic acid, acetate of lead, persulphate or perntrate of iron, or of calomel or of the saline cathartics. It is also customary to keep the patients very quiet while the bleeding is going on. I doubt if it be wise to be too anxious and energetic in the treatment of bleeding from the mucous membrane of the bronchi. The bleeding will regularly stop, no matter what is done. The frequent use of astringents disorders the stomach, the insistence of absolute quiet demoralizes the patient, the keeping the patients on a low diet unnecessarily reduces their strength.

## EMPHYSEMA.

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### INTERLOBULAR EMPHYSEMA.

*Definition.*—An accumulation of air in the connective tissue septa between the lobules of the lung.

*Etiology.*—Interlobular emphysema is, I think, most frequently seen with the broncho-pneumonia of young children. It may be caused by any violent efforts which require the abrupt introduction of a large quantity of air into the lungs, and its forcible retention therein by closure of the glottis. The efforts in parturition, defecation, raising weights, coughing, paroxysms of rage, excessive laughter, and hysterical convulsions have all been occasionally followed by rupture of the air-cells and interlobular emphysema.

*Morbid Anatomy.*—We find after death the interlobular septa infiltrated with air, with more or less compression of the parenchyma of the lung. The air may find its way from the lung into the mediastinum and thence into the connective tissue of the neck and the wall of the thorax.

*Symptoms.*—There seem to be no distinctive rational or physical symptoms belonging to interlobular emphysema. But in extreme cases it is said to have caused sudden death.

### VESICULAR EMPHYSEMA.

It is customary to speak of three forms of vesicular emphysema: Compensating emphysema, senile emphysema, and substantive emphysema.

1. COMPENSATING EMPHYSEMA.—If one lung, or a part of one lung, is so changed by disease that it can only partially perform its functions, the other lung becomes increased in size and its air-spaces are dilated. This change in the lung is a healthy, rather than a morbid one, and gives no symptoms of disease.

2. **SENILE EMPHYSEMA.**—This condition is often described as something different from substantive emphysema. It is said that, instead of there being an abnormal accumulation of air in the lungs, these organs are smaller and contain less air than normal. In consequence of atrophy of the alveolar walls the air-cells coalesce and form larger air-spaces. These, however, do not result from a dilatation of the alveoli, but from a gradual shrinkage and disappearance of the lung tissue.

I must confess that such descriptions of senile emphysema do not correspond with the lungs which I have seen. I believe that the anatomical conditions are the same in senile as in substantive emphysema, although the causation and clinical history are different.

3. **SUBSTANTIVE EMPHYSEMA.**—The definition of substantive emphysema usually given is that it is a morbid condition of the lungs, characterized by enlarged capacity of the air-cells, with atrophy of their walls, and obliteration of their capillaries.

I should define the disease, on the contrary, as a chronic interstitial inflammation of the lungs, with which more or less dilatation of the air-spaces is associated.

*Etiology.*—Laennec, who was the first to describe this lesion, gives the causation of emphysema as follows: chronic catarrh, plugging by mucus of small bronchi, consequent obstruction to the passage of air, conveyance by inspiration of air past the obstruction, failure of expiration to expel this air, accumulation of air in the air-spaces, dilatation of the air-spaces—the foundation of the disease, therefore, is bronchitis.

Louis denies this mechanism, because the symptoms of dilated air-cells are not preceded by catarrh, because habitual dyspnoea does not undergo permanent increase after acute catarrh, and because normal inspiration is not more powerful than expiration.

Dr. Williams maintains the catarrhal basis of Laennec, but supposes that while the air-cells communicating with plugged bronchi escape distention, those adjoining, and possessed of free communication with the trachea, dilate in consequence of the extra work and pressure thrown upon them.

Walshe says: The vesicular dilatation may be a primary or a secondary phenomenon; that is, it may occur independently of any acknowledged form of statical change within the chest, or it may supervene on some actual organic mischief. The dilatation may be the resultant of primary nutritive changes in the actual

walls of the enlarged vesicles, affecting both their statical and dynamic properties. Or these cells, being in their own nature healthy, may dilate through the extra strain thrown on them in consequence of the inaction of neighboring portions of lung.

It is frequently stated that playing on wind instruments or glass-blowing causes emphysema.

If, on the other hand, we believe that emphysema is a chronic interstitial inflammation of the lung, and that the dilatation of the air-spaces is not the primary or essential part of the morbid process, then we class emphysema with chronic endocarditis, endarteritis, and nephritis, and ascribe it to the same cause.

It is generally agreed that the disease is of more common occurrence in some families than in others.

The tradition has been handed down from one text-book to another that persons who have emphysema are less liable than are others to have tuberculosis of the lungs, or lobar pneumonia. This tradition seems to have no foundation in fact, tuberculosis and emphysema of the lung are frequently associated.

*Morbid Anatomy.*—Both lungs are regularly increased in size. The dilatation of the air-spaces may be so great as to be evident to the naked eye, or so moderate as not to be appreciable. When we examine minutely the lungs of a large number of persons who have, during life, suffered from the symptoms of emphysema, we find a very great variety. In some both the air-passages and air-vesicles are largely dilated; in some the air-passages alone are dilated, the air-vesicles remaining of normal size; in some neither the air-passages nor vesicles are appreciably dilated. We find also when we compare the lungs with the clinical histories which belong to them, that the most marked symptoms are often associated with very slight degrees of dilatation of the air-spaces, and it is evident that the severity of an emphysema is not to be measured by the degree of dilatation of the air-spaces.

The walls of the air-spaces are thickened in some parts of the lung, thinned in others. The epithelial cells which line the walls of the air-spaces are often increased in size and number. In the walls of the air-spaces are holes with sharp-cut edges. These holes are formed in the spaces between the capillaries, some are very minute, others are of large size. It is evident that these holes are not formed by the stretching of the air-spaces, for they are found in small air-spaces as well as in large ones. They constitute a curious and important part of the lesion. The septa

between the lobules, the connective tissue around the bronchi and blood-vessels, and the pulmonary pleura are often considerably thickened. Very frequently there are adhesions between the pulmonary and costal pleura. The mucous membrane of the larger bronchi is often thickened.

The blood-vessels of these lungs can be readily filled with an artificial injection. Neither arteries, capillaries, nor veins are obstructed or obliterated. But in the walls of dilated air-spaces, and in the walls of those in which there are holes the meshes of the capillary plexus are larger and the capillaries are farther apart from each other.

During life, however, in some cases of emphysema there is an obstruction to the passage of blood through the lungs, and consequently dilatation and hypertrophy of the right ventricle of the heart and venous congestion of the pia mater, stomach, small intestine, liver, spleen, and kidneys. These evidences of venous congestion often exist in cases in which the air-spaces are but very little dilated.

It is not often that we see a case of advanced emphysema after death without finding at the same time chronic endarteritis, endocarditis, or nephritis.

*Symptoms.*—Physical Signs. In the lesser degrees of emphysema there is no change in the shape of the thorax. In the more advanced cases there is a prominence of the upper part of the sternum and of the costal cartilages. In patients who have suffered much from dyspnœa the hypertrophy of the muscles which move the thorax contrasts with the general emaciation of the patients. In the cases in which there is great dilatation of the air-spaces the chest assumes the so-called barrel shape.

The percussion sound may remain unchanged for a considerable length of time. When it is changed the change is either to a rather dull note of wooden quality, or to exaggerated resonance of either vesicular, or vesiculo-tympanitic quality.

The respiratory murmur is feeble ; or there is feeble inspiration with longer, louder, low-pitched expiration ; or both inspiration and expiration may be exaggerated, loud, and high-pitched.

The pleuritic adhesions give more or less dulness on percussion. When the bronchi are contracted there is sibilant and sonorous breathing.

Rational Symptoms. There are many persons in whom substantive emphysema is developed and continues for many years

without giving rise to any symptoms, and yet, even in such persons, it is often possible to be pretty sure of the presence of the disease, because they are persons whose general condition and age are such as are usually associated with emphysema.

There are many persons in whom the associated chronic endocarditis or endarteritis, or nephritis, gives such marked symptoms that the emphysema passes unnoticed.

In some persons the emphysema gives after a time dyspnœa on exertion, but without bronchitis, or disturbance of the general health. These are persons past middle age, who do not consider themselves invalids, who, on the contrary, are often strong and robust, and in whom the emphysema is only an inconvenience.

In some persons the principal symptoms are those belonging to the associated acute and chronic bronchitis. The attacks of acute bronchitis may be mild, lasting for a few days or a few weeks, with cough, mucous expectoration, sometimes hæmoptysis, asthmatic breathing, and a febrile movement. Or they may be severe and last for several months, with continued cough, asthmatic breathing, fever, venous congestion, dropsy, and loss of flesh and strength. The chronic bronchitis continues year after year, better every summer and worse every winter. The patients have a cough with mucous, or muco-purulent expectoration, sometimes with small hæmoptyses. They are always a little short of breath when they exert themselves. After a time they have attacks of spasmodic asthma. In the unfavorable cases the dyspnœa on exertion becomes more constant and more decided, venous congestion and dropsy are established, and the patients lose flesh and strength.

In some persons the prominent feature in the disease is the liability to attacks of spasmodic asthma, which often are frequently repeated and of long duration. These attacks are sometimes due to contraction of the bronchi, and then we get sibilant and sonorous breathing; or they are due to contraction of the arteries belonging to the aortic system and there is a radial pulse of increased tension; or they are due to contraction of the branches of the pulmonary artery and then there is neither sibilant and sonorous breathing, nor a pulse of high tension.

In some persons the principal symptom is the constant dyspnœa. The difficult breathing is at first only developed by exertion, later it becomes more constant and is made worse by slight

exertion, by indigestion, and by bronchitis. Finally, in the bad cases, the dyspnœa is constant and distressing. The patients constantly feel the need of air and are always overusing the muscles of respiration in order to satisfy this need. General venous congestion is gradually established as well as cyanosis of the skin, clubbing of the fingers, congestion of the stomach, small intestine, liver, and kidneys; dilatation and hypertrophy of the right ventricle of the heart and general dropsy. The nutrition of the patients suffers and they become emaciated, feeble, and anæmic. It is not easy to tell how much of this dyspnœa depends upon the anatomical changes in the lungs, how much upon contraction of the branches of the pulmonary artery, and how much upon contraction of the arteries belonging to the aortic system. There are rare and fatal cases in which there are no pulmonary symptoms. The patients lose flesh and strength and become anæmic without any evident cause for their ill-health. After going on in this way for some time they begin to have attacks of contraction of the arteries with headache, sleeplessness, delirium, stupor, muscular twitchings, and vomiting, or a dyspnœa like that seen in chronic nephritis. They die within a few months after they have begun to have the attacks of contraction of the arteries.

In some persons, after emphysema has existed for some time with more or less marked symptoms, chronic miliary tuberculosis is slowly established.

Emphysema by itself proves fatal only in a moderate number of cases. Death is usually due to some complicating or intercurrent disease.

*Treatment.*—The conditions which call for treatment are: The morbid condition of the lung, the loss of nutrition, the bronchitis, the constant dyspnœa and the spasmodic dyspnœa; the contraction of the arteries, and the venous congestion.

The emphysema is favorably affected by an out-of-door life in a suitable climate; by abstinence from alcohol, tobacco, sugars, and starches; by the use of fats and by overfeeding with the stomach-tube; and by methodical inhalations of compressed air. All of these measures are also of service in improving the nutrition of the patients and in helping them to get rid of chronic bronchitis.

The constant dyspnœa is due to the changes in the lungs, and is then to be treated by the same means which are used to con-

trol the emphysema; or it is due to the complicating contraction of the arteries, and is then to be treated by the drugs which dilate the arteries—nitro-glycerine, potassium iodide, or chloral hydrate.

The attacks of spasmodic dyspnœa are due to :

(a) Spasmodic contraction of the muscular coat of the bronchi. This can be relieved by the inhalation of the fumes of stramonium, nitrate of potash, chloroform, or ether; by the administration of chloral hydrate, potassium iodide, belladonna, or opium.

(b) Congestion of the walls of the bronchi. This can be relieved by drugs which increase the production of mucus, such as lobelia and grindelia robusta; by drugs which stimulate the heart, such as caffeine, convallaria, and digitalis; or by the application of dry cups to the walls of the chest.

(c) Contraction of the small arteries. This can be relieved by the drugs which dilate the arteries, such as nitrite of amyl, nitro-glycerine, chloral hydrate, potassium iodide, or opium.

(d) Inflammation of the nasal passages. This is to be treated by local applications.

#### ACTINOMYCOSIS OF THE LUNG.

The following account of actinomycosis of the lung is taken from a compilation of thirty-four cases made by Dr. Hodenpyl. My own experience is limited to two cases.

*Definition.*—Pulmonary actinomycosis is a chronic infectious disease of the lungs dependent upon the presence of actinomyces.

*Etiology.*—Information concerning the characters and causation of actinomycosis in general are given in the article on that disease. So far as the lungs are concerned the living germ seems to be inhaled into the bronchi. The majority of the cases were in young adults; the youngest patient was nine years old, the oldest sixty-three.

*Morbid Anatomy.*—The lesions are unilateral in about the proportion of three to one. They may be classified into two groups.

1. There are cases with the symptoms of chronic general bronchitis with the germ present in the sputum, but in which no autopsy is made. Whether in these cases there is no lesion but that of chronic bronchitis we cannot certainly tell.



2. There is a broncho-pneumonia of a peculiar type, which involves part of a lobe, or an entire lung. The large bronchi are coated with muco-pus. The small bronchi contain pus, their walls are thickened, they are surrounded by zones of peri-bronchitic pneumonia. In some of the small bronchi there are growths of new connective tissue partly filling them. In the peri-bronchitic zones of pneumonia the walls of the air-spaces are thickened and their cavities filled with new connective tissue. Between these zones is a diffuse hepatization of ordinary exudative type. There are adhesions between the pulmonary and costal pleura.

There are often, in addition, sacculated collections of pus in the pleural cavity, which may perforate through the skin, or through the diaphragm. The ribs, sternum, or vertebrae may be eroded. The opposite lung, the pericardium, or the heart may become involved. There may be secondary inflammations of the abdominal organs, or of the brain. In one case the inflammation penetrated the portal vein and there were metastases all over the body.

*Symptoms.*—A febrile movement is present in nearly all the cases. Usually it is one of the first symptoms, but sometimes it does not come on until later in the course of the disease.

Cough is regularly the first symptom, and continues throughout the disease. It is accompanied with an abundant mucopurulent, often fetid, expectoration, and sometimes contains actinomyces. Hæmoptyses were not observed, although the sputa were sometimes stained with blood. The patients lose flesh and strength, at first slowly, later, as abscesses are formed and septic poisoning established, they run down more rapidly.

The physical signs are those of bronchitis, of broncho-pneumonia, of phthisis, or of empyema.

The average duration of the disease is ten months; the shortest case lasted four months, and the longest was still living after a duration of several years.

Of the thirty-four cases all died except two.

*Diagnosis.*—The disease is liable to be confounded with fetid bronchitis, gangrene of the lung, broncho-pneumonia, or pulmonary phthisis. The only positive diagnostic symptom seems to be the presence of the actinomyces in the sputa or in the pus from the pleura.

*Treatment.*—There seems to be no way of directly improving this disease of the lungs.

#### MALIGNANT GROWTHS IN THE LUNGS.

We include under this head the primary and secondary tumors formed in the lungs which belong to the classes of carcinoma, sarcoma, and lymphoma.

*Morbid Anatomy.*—The carcinomata of the lung are either secondary or primary. The secondary tumors follow the anatomical type of the primary tumor. The primary tumors consist of a stroma enclosing spaces lined with cylindrical epithelium, the growth apparently beginning in the small bronchi. Whether the tumors are primary or secondary we find that they may be developed in such a way as to compress the bronchi, or be associated with exudative and productive inflammation in such a way as to consolidate large portions of the lung; or be associated with suppurative and destructive inflammation in such a way that abscesses are formed; or involve the pleura so that large collections of serum are formed in the pleural cavities.

The sarcomata of the lung are secondary tumors. They usually are in the form of nodules scattered through the lung, or of tumors which compress the bronchi, or of tumors in the pleura with serum in the pleural cavities.

The lymphomata begin in the bronchial glands and infiltrate the lungs from the root outward, following the track of the bronchi or of the interlobular septa.

*Symptoms.*—While in some cases the symptoms are obscure, in others they are well defined. They are apt to follow one of three types:

1. The most marked symptom is dyspnoea due to pressure on the bronchi. The dyspnoea is developed slowly and is brought on by exertion. It becomes more and more distressing until finally the patient can hardly move at all without bringing on the bad breathing.

The physical signs are either tubular breathing, or diminished breathing over the lung corresponding to the compressed bronchi. The patients lose flesh and strength, at first slowly, later very rapidly.

2. The symptoms are those of a chronic inflammation of the lung. The patients have cough, dyspnoea, muco-purulent or

bloody expectoration, a febrile movement, pains in the chest, the physical signs of bronchitis and of consolidation of the lung, and gradual loss of flesh and strength.

3. The symptoms are those of a pleurisy with effusion, but it is a pleurisy which does not improve under treatment. The serum is apt to be blood stained, but is not always so, it reaccumulates after it has been drawn off, the patients steadily lose flesh and strength, or there may be a combination of the signs of consolidation of the lung with that of fluid in the pleura.



## THE HEART.

IN examining any person who is suspected of having a disease of the heart we follow a certain routine in the physical examination. We determine the size of the heart, the character of its impulse, the rhythm of its contractions and dilatations, the character of the heart-sounds, and the presence or absence of murmurs.

The size of the heart is made out by simple, or by auscultatory percussion, and by locating the apex-beat. We determine the upper, the left, and the right edges, and the apex-beat.

The upper border of the heart should be at the third left costal cartilage. The left border is at the left nipple. The right border is one inch to the right of the sternum. The apex-beat is in the fifth interspace, midway between the left edge of the sternum and the nipple, three and one-fourth inches from the middle of the sternum. A small portion of the anterior surface of the pericardium, usually corresponding to the fourth left interspace and the fifth left cartilage, is uncovered by the lungs, and gives more decided dulness on percussion than the rest of the precordial area.

It is comparatively easy in this way to make out any increase in the size of the heart, but a diminution in its size is much more difficult to determine.

The apex-beat can usually be felt by the hand, but in many healthy persons does not communicate a perceptible shock to the chest-wall. Its position must then be determined by the stethoscope. The force of the impulse is temporarily increased by muscular exertion, by rapid respiration, by digestion, and by mental emotions. It is decreased by any disease which impairs nutrition.

Myocarditis, degeneration of the wall of the heart, an excess of fat about the heart, and fluid in the pericardial sac diminish

the force of the impulse. There are often cases of abnormally feeble impulse for which it is difficult to account.

The force of the impulse is increased in some of the cardiac neuroses, with hypertrophy of the ventricles, with contraction of the arteries, and with some of the acute inflammations of the pericardium and endocardium.

Dilatation of the ventricles changes the character of the impulse; instead of a circumscribed apex-beat there is a diffuse, heaving impulse over the whole precordial region.

The rhythm of the alternate contractions and dilatations of the heart should be perfectly regular. In some healthy persons, however, there is a regular intermission of the ventricular systole. The same thing may occur in the tubercular meningitis of children. In some persons the heart-sounds are reduplicated; either the systolic, or the diastolic sound, or both, may be reduplicated; this is said to be due to a want of synchronism between the action of the two sides of the heart.

The heart's action becomes irregular with valvular disease, dilatation and hypertrophy of the ventricles, fatty degeneration of the wall of the heart, myocarditis, pericarditis, and the cardiac neuroses.

*The Normal Heart-sounds.*—The contraction and dilatation of the cavities of the heart are accompanied with certain sounds. The first sound is synchronous with the systole of the ventricles, the apex-beat, and the closure of the auriculo-ventricular valves; it is loudest at the apex. After the first sound is a short interval of silence, the post-systolic silence. The second sound is synchronous with the closure of the aorta and pulmonary valves; it is most distinct over the third cartilage and the middle of the sternum. After the second sound is another interval of silence, the post-diastolic silence. If the period of an entire revolution of the heart from the beginning of one first sound to the beginning of the next first sound be divided into ten equal parts, the duration of the several periods of sound and silence will be as follows: First sound, 4; first silence, 1; second sound, 2; second silence, 3.

When we listen to the heart-sounds we notice the loudness, the distinctness, and the quality.

Murmurs are pericardial or endocardial.

Pericardial murmurs are produced by the rubbing of opposed surfaces of the pericardium coated with fibrin or with adhesions.

The sound may be of a rubbing, grating, creaking, squeaking, or whistling character ; or it may be soft and blowing like an endocardial murmur. The sound may be very loud, or so faint as to be hardly audible. Such murmurs are heard with the first or second sounds, or with both ; less frequently they are pre-systolic ; but they are not always exactly synchronous with the heart-sounds.

There is no fixed point of intensity for pericardial murmurs, but they are most frequently heard over the base. Usually the sound seems to be superficial. Such murmurs are distinguished from endocardial murmurs by their quality, their superficial character, their limitation to a small area, their changeableness in position and intensity from hour to hour, their greater intensity when the patient leans forward or fills the lung, their greater intensity if we press on the chest-wall, their want of synchronism with the heart-sounds. But in some cases it is hardly possible to distinguish between pericardial and endocardial murmurs.

Endocardial murmurs are produced by :

(a) Change in the valves, by which they are rendered rough, stenosed, or insufficient.

(b) Ventricular lesions. Inflammation of the endocardium or of the chordæ tendinæ, abnormal tendinous cords extending across the ventricles, or thrombi in the ventricles. Ventricular murmurs are systolic, their point of maximum intensity is at the apex ; they are not transmitted.

(c) Dilatation of the right auricle and the right ventricle. Such dilatations disturb the circulation in the pulmonary artery and aorta. They cause a systolic murmur, with its point of maximum intensity at the second left intercostal space, or at the apex. The murmur can be heard in some persons at all times ; in others, only after muscular exertion ; in others, only when they are anæmic.

#### PERICARDITIS.

*Lesions.*—The inflammations of the pericardium resemble those of the pleura.

They begin acutely or subacutely, and then run an acute, subacute, or chronic course. There is a greater disposition to bleeding from the inflamed surfaces than is the case in pleurisy. The inflammation regularly begins at the base of the heart and then extends over the greater part of the pericardium.

We distinguish :

1. *Pericarditis with the Production of Fibrin*.—The pericardium is congested, or even studded with minute hemorrhages. Its surface is coated with a thin film of fibrin, or with large, rough masses, which glue together the opposed surfaces of the pericardium. If the inflammation is protracted the pericardium itself becomes thickened and infiltrated with cells, and the wall of the heart may also undergo inflammatory changes.

If the patient recovers the fibrin is absorbed and the pericardium returns to its normal condition ; or there is a growth of new connective tissue, which forms permanent thickenings and adhesions.

2. *Pericarditis with the Production of Fibrin and Serum*.—The pericardium is coated with fibrin and there is an effusion of serum in the pericardial sac. The serum accumulates at first between the floor of the pericardium and the lower surface of the heart, as it increases it distends the pericardial sac in all directions, pushing the heart upward and forward. The pericardial sac may be so much distended as to compress the trachea, the left bronchus, the œsophagus, or the aorta. If the patients recover, the serum and fibrin are absorbed, but permanent thickenings and adhesions of connective tissue are left.

3. *Pericarditis with the Production of Fibrin, Serum, and Pus*.—This variety may have the purulent character from the outset, or it may begin as one of the forms just described, and afterward assume the purulent character. The inflammation is apt to run a chronic course. The pericardial sac contains a large quantity of purulent serum ; the pericardium is coated with fibrin, thickened and infiltrated with cells ; the walls of the heart may be the seat of interstitial myocarditis. The trachea, left bronchus, œsophagus, or aorta may be compressed. The serum may be absorbed, and the fibrin and pus undergo cheesy degeneration, or there may be a deposition of the salts of lime in the thickened pericardium.

*Causes*.—Altogether the most frequent cause of pericarditis is acute articular rheumatism. With scarlatina and the other infectious diseases it is not uncommon. It complicates chronic nephritis, and sometimes is the first symptom to call attention to the nephritis. A double pleurisy is regularly accompanied with pericarditis, while with a single pleurisy this is not often the case. Lobar pneumonia is often complicated by pericarditis. This



happens more often in some years than others. There are winters during which a considerable number of deaths from pneumonia seem to be due to the pericarditis. Primary pericarditis seldom occurs.

*Symptoms.*—In pericarditis with the production of fibrin alone the regular physical signs are the friction-sound and the increased frequency of the heart's action. But with large quantities of fibrin the heart dulness is symmetrically enlarged.

In pericarditis with the effusion of serous or purulent fluid the physical signs vary with the quantity of fluid. With small quantities of fluid the extent of the precordial dulness is unchanged, but the dulness approaches flatness.

With distention of the pericardial sac by serum the area of precordial dulness is enlarged in all directions. The anterior wall of the chest may be bulged out. The apex-beat is carried upward. Before the appearance of the fluid and after its absorption there may be a friction-sound. The physical signs due to the pericarditis may be complicated by murmurs due to a pre-existing endocarditis.

The rational symptoms of pericarditis are pain, rapid, feeble, and irregular heart-action, rapid and oppressed breathing, venous congestion of the skin and viscera, dysphagia, a febrile movement, and prostration.

The cases with exudation of fibrin alone give the fewest and least severe symptoms; those with large exudations of serum give very marked symptoms; those with serum, fibrin, and pus are the worst.

In many of the milder cases it is only by the physical signs that we recognize the existence of the disease.

Sometimes, however, the symptoms of a dry pericarditis are so marked as to mask those of a lobar pneumonia.

The cases with large effusions of fluid do not seem to be as common in the United States as in some other countries.

*The prognosis* in the milder cases depends more upon the primary disease than upon the pericarditis. With the large serous and purulent effusions, however, the pericarditis becomes in itself a dangerous lesion.

*Treatment.*—For a rheumatic pericarditis the treatment of the rheumatism with salicylate of soda, etc., is a matter of much importance. With pneumonia and with double pleurisy the treatment of these conditions is necessary.

For the pericarditis, dry cups, blisters, or continuous cold over the precordial region, are of service. The feeble and disturbed heart-action requires cardiac stimulants. If there is a considerable quantity of fluid in the pericardial sac we use cathartics or diuretics, or draw off the fluid with the aspirator. The point of election at which to insert the trocar is the fifth left interspace, about two or three and a half inches from the sternum.

#### ENDOCARDITIS.

The endocardium is a connective-tissue membrane which lines the cavities of the heart and forms the larger part of the valves. It is a thin, dense, and elastic membrane, containing but few blood-vessels. It becomes the seat of productive rather than of exudative inflammation. During foetal life the endocardium of the right heart is inflamed; after birth usually the endocardium of the left heart. The valves are the portion of the endocardium most frequently affected, but that which lines the auricles and ventricles and covers the papillary muscles may be inflamed independently.

We distinguish:

Acute endocarditis.

Malignant endocarditis.

Chronic endocarditis.

#### ACUTE ENDOCARDITIS.

*Lesions.*—1. The valves are swollen and succulent; the basement-substance and cells are swollen; the surface of the valves remains smooth. As the valves are swollen they are also shortened and insufficient.

2. The valves are not only swollen but there is an extensive growth of their connective-tissue cells, resulting in the formation of ulcers and of vegetations. On the roughened surfaces of the valves fibrin from the blood coagulates in considerable masses. Portions of the vegetations may become detached, pass into the aorta, and become lodged in the smaller arteries as emboli.

*Causes.*—Acute endocarditis is usually a complication of rheumatism, of scarlatina, or of chorea.

*Symptoms.*—The physical signs are due to the insufficiency and

roughening of the valves. So we have a mitral systolic, or aortic diastolic, or double aortic murmur.

The rational symptoms are :

Fever, prostration, disturbed action of the heart with consequent changes in the circulation, and symptoms due to embolism. The disturbed heart-action follows one of two types :

1. The heart's action is violent, tumultuous, and irregular ; there is very marked venous congestion of different parts of the body.

The congestion of the pia mater produces delirium and stupor.

The congestion of the lungs gives labored breathing, and may go on to inflammation and consolidation of parts of the lungs.

The congestion of the pleura produces hydrothorax ; that of the stomach, vomiting ; that of the liver, enlargement of that organ ; that of the peritoneum, ascites ; that of the subcutaneous connective tissue, dropsy.

The congestion of the kidneys is attended with a diminished quantity of urine, which contains a little albumin and a few casts. If the urine is scanty for a long time the patients become anæmic and develop the symptoms of chronic uræmia.

2. The heart's action is feeble and rapid. There is not as much disposition to venous congestion, but the patients suffer from dyspnœa, they are very feeble, they are liable to attacks of syncope.

The emboli which give the most marked symptoms are those which become lodged in the cerebral arteries.

A large number of cases of acute endocarditis recover, but very few without permanent changes in the valves.

In some the valves, although rendered insufficient, undergo no further changes, and the patients do well.

In some the valves, after remaining unchanged for years, finally become the seat of chronic endocarditis.

In some the acute endocarditis passes into the chronic form, and the patients grow worse with but little interruption.

*Treatment.*—The principal objects of treatment are the cure of the rheumatism and the relief of the bad heart-action with the consequent disturbances of circulation. For the rheumatism the most efficient remedies are the salicylate of soda, the oil of wintergreen, the alkalies, antipyrine, and phenacetine.

If the heart's action is exaggerated, tumultuous, and irregular, with excessive venous congestion, we employ venesection, wet cups or leeches, purging or sweating. Chloral hydrate, amyl nitrite, opium, and potassium iodide are often of much service.

If the heart's action is feeble and rapid we need the cardiac stimulants—digitalis, convallaria, caffeine, and alcohol.

#### MALIGNANT ENDOCARDITIS.

*Lesions.*—Either the mitral, the aortic, or the tricuspid valve, the endocardium of the left auricle, or of the right ventricle may be inflamed. The inflamed endocardium is covered with a thin or thick layer of soft material composed of fibrin and micrococci, usually the ordinary streptococci and staphylococci of supuration or the diplococcus pneumoniae. The endocardium beneath is swollen, infiltrated with cells, often ulcerated. If the malignant endocarditis affects a heart already the seat of chronic endocarditis, the old lesions persist.

Portions of the soft matter on the inflamed endocardium often become detached and are lodged as emboli in the arteries of the brain, lungs, heart, spleen, and kidneys. These emboli are infectious and set up an inflammation of the surrounding tissues. So there may be complicating meningitis, pericarditis, bronchitis, pneumonia, or synovitis. The liver, spleen, and kidneys are swollen and their cells degenerated.

*Causes.*—In order to have a malignant endocarditis, it is necessary to have some ordinary cause of endocarditis combined with an infection by pathogenic bacteria. The bacteria found most frequently are the streptococci, staphylococci and diplococcus pneumoniae. Such an endocarditis may attack a normal heart, or one in which the lesions of chronic endocarditis already exist.

*Symptoms.*—There are two very noticeable features about the disease.

1. The very great predominance of the infectious symptoms over the cardiac symptoms. Often the heart's action is regular, there are no disturbances of circulation, not much of a murmur, very little to call attention to the heart. On the other hand the evidences of systemic poisoning—the fever, cerebral symptoms, and prostration are very marked.

2. When this form of endocarditis attacks a heart already the

seat of chronic endocarditis, two distinct histories become continuous. There is the ordinary history of a chronic endocarditis extending over months or years, and then at some particular time are added the symptoms of infection.

*Symptoms.*—The disease begins with chills and a febrile movement. There is loss of appetite and sometimes vomiting; the breathing is rapid. The heart's action may not be very much disturbed; or it may be feeble, rapid, and irregular. The temperature continues high— $103^{\circ}$  F. to  $107^{\circ}$  F.—but with occasional rapid falls. Cerebral symptoms are soon developed—restlessness, sleeplessness, delirium, and stupor. There is a heart murmur if the endocarditis is sufficient to produce insufficiency or roughening of the valves. The urine contains some albumin; the joints may be inflamed; there may be convulsions or paralyses from embolism of the larger cerebral arteries. There may be bleeding from the mucous membranes and into the skin.

The patients pass into a typhoid condition and die, most at the end of seven to ten days, a few within three days, some not until after three to five weeks.

The whole picture is that of an infectious disease, with more or less pronounced evidence of the cardiac lesion.

*The diagnosis* of malignant endocarditis is a very difficult one. The cases resemble very closely meningitis, general tuberculosis, typhoid fever, and septic poisoning.

*Treatment.*—There seems to be no treatment for this disease.

#### CHRONIC ENDOCARDITIS.

*Lesions.*—The inflammation involves most frequently the mitral and aortic valves, less often the tricuspid and pulmonary valves and the endocardium of the ventricles and auricles. There are two forms of chronic endocarditis.

1. The endocardium is thick, dense; there may be little, beaded vegetations on its surface; it may be infiltrated with the salts of lime. But its surface remains smooth, and thrombi are not formed on it. Such an endocarditis renders the valves rigid, insufficient, or stenosed.

2. The endocardium is thickened, there is a growth of cells producing vegetations and ulcers, the surface of the endocardium is roughened, and thrombi are formed on it. In such an

inflamed endocardium there may also be a deposition of the salts of lime.

When the valves are affected by either of these forms of endocarditis they are rendered insufficient, or stenosed. These changes are followed by dilatation of the ventricles, by hypertrophy of their walls, and by disturbances of the circulation of the blood throughout the body.

With chronic endocarditis are often associated myocarditis, endarteritis, contraction of the arteries, emphysema, and chronic nephritis.

*Causes.*—Chronic endocarditis may succeed acute endocarditis, either immediately or after a long interval. It may be caused by rheumatism, gout, or syphilis. It very often is a primary lesion. It is rarely due to the rupture of a valve by external violence.

*The symptoms* depend on :

The endocarditis.

The dilatation and hypertrophy.

The character of the heart's action.

The presence of thrombi on the endocardium.

The myocarditis.

The endarteritis.

The contraction of the arteries.

The emphysema.

The chronic nephritis.

The general venous congestion.

1. *The Endocarditis.*—The inflammation may last for months or years, cease to be active, and no further changes be produced in the valves ; or it may continue slowly and with intermissions up to the time of the patient's death, the valves becoming more and more damaged, and the inflammation extending to other valves ; or the inflammation may be more active, the endocardium more rapidly and profoundly changed, with a febrile movement and loss of nutrition.

The changes in the endocardium, due to the inflammation, may produce cardiac murmurs.

Aortic stenosis gives a murmur with the first sound, and during the first silence. The murmur is loudest at mid-sternum, opposite the third interspace. It is transmitted upward and to the right. It may be heard behind on the left side of the second, third, and fourth dorsal vertebræ. It may be loudest at the lower end of the sternum, or in the second interspace, on the

right or left side. There may be well-marked aortic stenosis with no murmur.

Aortic insufficiency gives a murmur with the second sound, loudest at mid-sternum opposite the third interspace. It is transmitted down the sternum, and upward to the right. It may be loudest at the lower end of the sternum, in the left fourth interspace, or at the apex. It is a very constant murmur.

Mitral stenosis gives a presystolic, or systolic murmur, or both. The presystolic murmur is heard just before the first sound. It is loudest at the apex and is usually circumscribed. The systolic murmur is loudest at the apex and is transmitted to the left. The murmurs of mitral stenosis are very inconstant, and often absent altogether.

Mitral insufficiency gives a systolic murmur, loudest at the apex, and transmitted to the left. This murmur is nearly always a constant one.

Ventricular murmurs are produced by roughening of the ventricular endocardium, or thrombi in the ventricles. They are systolic, loudest at the apex, and circumscribed. They are not constant.

2. *Dilatation and Hypertrophy of the Ventricles.*—Dilatation alone of the ventricles adds to the dangers of the endocarditis. Hypertrophy alone seems often to be of benefit. Hypertrophy and dilatation together, if not excessively developed, may also be of benefit. A heart which remains of normal size, or which becomes small when associated with advanced valvular changes, is apt to act badly.

Aortic stenosis is regularly followed by dilatation and hypertrophy, first of the left ventricle, then of the right.

Aortic insufficiency is followed by dilatation and hypertrophy of the left ventricle.

Mitral stenosis should be followed by dilatation and hypertrophy of the left auricle and the right ventricle. But there may be dilatation alone of either or of both ventricles; or dilatation and hypertrophy of the left ventricle; or the entire heart may be small.

Mitral insufficiency is followed by dilatation alone, or dilatation and hypertrophy of the left ventricle.

3. *The Character of the Heart's Action.*—Disturbances of the heart's action seem to depend upon the changes in the valves

and walls of the heart; upon contraction of the arteries; and upon unknown causes.

The heart's action may be rapid, slow, feeble, forcible, intermittent, irregular, or tumultuous. The contractions of the heart may be so feeble that there are more apex-beats than arterial pulsations.

Aortic and mitral stenosis, without contraction of the arteries, give a feeble and irregular pulse; with contraction of the arteries, the pulse is tense, and the heart-action often laboring. Aortic stenosis may give more ventricular contractions than heart-beats.

Aortic insufficiency gives an exaggerated heart action, with dilated arteries, full of blood, but soft.

Abnormal sensations referred to the heart seem to depend upon its disturbed action.

There may be actual pain, referred to the heart and the left shoulder; the pain constant, produced by exertion, or paroxysmal. Or there are feelings of constriction, or of displacement of the heart.

4. *Thrombi Attached to the Endocardium*.—These interfere with the heart's action. They may obstruct the orifices of the heart; fragments of them lodged in the cerebral arteries produce convulsions, paralysis, and aphasia.

5. *Myocarditis*.—Interstitial myocarditis, degeneration of the heart-muscle, and disease of the coronary arteries are each one capable of rendering the heart's action feeble and irregular, of producing attacks of angina pectoris, and of causing sudden death.

6. *Chronic endarteritis* affecting many of the arteries adds to the difficulties of the circulation. If it involves the cerebral arteries the characteristic symptoms of cerebral endarteritis may be developed.

7. *Pulmonary emphysema* is a frequent complication, and adds much to the dangers of the endocarditis.

8. *Venous congestion* takes a very large share in producing the symptoms of chronic endocarditis. It is due to the valvular lesions, the dilatation of the ventricles, the disturbed heart-action, the contracted arteries, and the complicating emphysema or endarteritis.

Congestion of the pia mater produces delirium, stupor, and convulsions.



Congestion of the lungs produces chronic bronchitis, hæmoptyses, the pneumonia of heart disease, œdema of the lungs, and hydrothorax.

Congestion of the stomach produces pain, gastric dyspepsia, vomiting, and vomiting of blood.

Congestion of the small intestine produces loss of flesh and strength.

The general congestion of the body gives subcutaneous dropsy and dropsy of the serous cavities.

The kidneys may be the seat of chronic congestion, of chronic degeneration, or of chronic nephritis.

With chronic congestion of the kidney the quantity of urine is diminished, its specific gravity is high, there is little or no albumin, the quantity of urea to the ounce of urine is sufficient. This change in the kidney seems to add but little to the symptoms.

The chronic congestion, if protracted, is succeeded by a chronic nephritis. The specific gravity of the urine falls, the quantity of urea is diminished, and albumin and casts make their appearance.

With chronic degeneration of the kidney the quantity of the urine is diminished, the specific gravity and the proportion of urea are unchanged, there is a moderate quantity of albumin and a few casts. If the excretion of urine is scanty for a long time the patients may pass into the condition of chronic uræmia.

The chronic nephritis is diffuse and most frequently follows one of two types.

1. There is an abundant exudation of serum from the vessels of the kidneys. The urine is of low specific gravity and contains large quantities of albumin. The patients are anæmic, dropsical, and suffer from chronic uræmia.

2. There is little or no exudation from the blood-vessels of the kidneys, but there is an extensive growth of interstitial connective tissue. The urine is of low specific gravity, contains little urea, and little or no albumin. The patients are liable to attacks of acute uræmia, with contracted arteries.

*Course of the Disease.*—The disease may succeed one or more attacks of acute endocarditis, or it may be chronic from the outset.

In the chronic cases the symptoms are usually not developed until the disease has existed for some time. Then first one or two symptoms appear and others are added later.

The first symptom may be :

Dyspnœa on exertion.

Anæmia and emaciation.

Cough and expectoration.

Cardiac palpitation and pain.

Stomach symptoms.

Dropsy.

Contraction of the arteries.

It sometimes happens that the patients go on for a long time with an endocarditis, but without symptoms. Then suddenly, with a pericarditis, a pleurisy, a bronchitis, or without discoverable cause, they develop a most distressing and urgent dyspnœa with contracted arteries. This dyspnœa continues and the patients die within a few days or weeks.

The endocarditis may stop at any time. It may go on slowly, or quickly ; it may be complicated by the diseases already mentioned.

The bad cases die with dyspnœa ; or feeble and emaciated ; or with dropsy ; or with cerebral symptoms ; or they die suddenly.

*The prognosis depends upon :*

The extent and activity of the endocarditis.

The condition of the ventricles.

The character of the heart's action.

The presence or absence of contraction of the arteries.

*Treatment.*—The principal indications for treatment are to check the progress of the endocarditis, to regulate the action of the heart and the condition of the arteries, and to alleviate symptoms.

To check the progress of the endocarditis we put the patient on a diet of fats, meat, fruits, and vegetables ; we prohibit the use of alcohol and tobacco, and we insist on a life passed in the open air.

To regulate the action of the heart and the condition of the arteries. If the arteries are contracted we employ nitrite of amyl, nitroglycerine, potassium iodide, chloral hydrate, or opium.

If the heart's action is feeble we use digitalis, strophanthus, caffeine, or convallaria. If the heart's action is too forcible we use continuous cold over the heart, a nitric-acid issue, aconite, veratrum viride, or barium chloride.

With aortic, or mitral, stenosis and a feeble heart it may be necessary to confine the patients to bed.

With many forms of endocarditis the patients do best if they are allowed to follow their ordinary mode of life, avoiding sudden and violent exertion.

In a certain number of cases very excellent results are obtained by regular hill-climbing.

#### CHRONIC ENDOCARDITIS WITH FEVER.

In the course of any chronic endocarditis there may be loss of nutrition, and on some days a rise of temperature. But there are some cases of chronic endocarditis in which the rise of temperature and the loss of nutrition are the prominent symptoms, while there is but little disturbance of the circulation. The behavior of the cases is such as to remind one of malignant endocarditis and to give the impression that the symptoms depend rather on bacterial poisoning than on the cardiac lesion.

*Lesions.*—Either the aortic or the mitral valves are affected. There is a growth of cells in the endocardium with more or less ulceration and the formation of vegetations. The bacteriology has not been worked up.

*Symptoms.*—The most noticeable symptom is the fever. In most of the cases there is every day an afternoon temperature of from 100° to 104° F., while the morning temperature is from 97° to 99° F. With the fever there may be chilliness and sweating. Less frequently the fever comes on every other day. The whole behavior of the fever is such as to make one think of malarial poisoning. It is said that in some cases the endocarditis is preceded by a true malarial fever.

The patients are at first not confined to bed, but gradually lose flesh and strength. There is a cardiac murmur, but no other cardiac symptoms. There are often, however, days and weeks of considerable improvement and less fever. But then the fever comes on again and the patients get worse. They continue in this way for from six to twelve months, and finally die in a condition of extreme feebleness and emaciation.

#### HYPERTROPHY AND DILATATION OF THE HEART WITHOUT ENDOCARDITIS.

##### I. From excessive and prolonged muscular exertion.

The left ventricle alone, or both ventricles, are hypertrophied or dilated, or both dilated and hypertrophied.

To produce these changes in the heart the muscular exertion must be excessive and prolonged, so we find them most frequently in soldiers, athletes, and laborers.

In the milder cases there is only hypertrophy of the left ventricle. The patient has the feelings of pain, constriction, or displacement of the heart, with rapid heart-action.

These patients usually do well. It is only necessary to make them lead a regular life and sometimes to take digitalis.

In the more severe cases there is dilatation alone, or dilatation with hypertrophy. There are pains over the heart, rapid and feeble heart-action, dyspnœa on exertion, and loss of flesh and strength. These cases are always serious, and not all of them recover.

It is necessary to keep the patients in bed, and to use cardiac stimulants.

## II. From disease of the lungs.

Emphysema, chronic phthisis, interstitial pneumonia, pleurisy, and deformities of the chest may produce dilatation, first of the right ventricle, and afterward of the left.

## III. Exophthalmic goitre, or prolonged palpitation of the heart, may be followed by hypertrophy of the left ventricle.

## IV. Acute or chronic nephritis, chronic inflammation of the aortic system of arteries, and congenital narrowing of the aorta may produce hypertrophy of the left ventricle.

## V. Simple anæmia may be attended with temporary dilatation and hypertrophy of the left ventricle, or of the right auricle and ventricle.

## VI. There is an important group of cases of dilatation of the ventricles without hypertrophy, of which the causation is very obscure.

Usually both ventricles are dilated, but sometimes only the left. The valves are normal, but their orifices may be so large that they are insufficient. The walls of the ventricles are normal, or the muscular fibres are degenerated, or there is a growth of cells between the fibres. Thrombi may be formed in the cavities of the ventricles. Venous congestion of the viscera is soon established, dropsy somewhat later.

Of the causes of this heart-lesion we are ignorant. It is often enough developed in young and healthy adults. In some cases there is a distinct history of sudden muscular exertion ; in others, no exciting cause can be discovered.

In the more acute cases the invasion of the symptoms is sudden.

The patients complain of pain in the epigastrium, they become unconscious, and they vomit ; or the first symptom is urgent cardiac dyspnœa. After this the heart's action is rapid, irregular, and feeble. The physical signs are those of dilatation of the heart. There is constant and distressing dyspnœa, there may be cough and hæmoptyses, the patients lose flesh and strength, and die at the end of a few weeks.

In the more chronic cases the invasion is less sudden and decided. The patients suffer from rapid, feeble, and irregular heart-action, dyspnœa, cough, and hæmoptyses, marked dropsy, and loss of flesh and strength. They may live for several months.

The treatment of these cases is eminently unsatisfactory. We are unable to improve the action of the heart, and the best that we can do is to palliate the patient's sufferings with opium.

#### THE FATTY HEART.

1. There is an accumulation of fat beneath the pericardium. This occurs in persons who also have accumulations of fat in other parts of the body.

The heart's action is often feeble, and there is dyspnœa on exertion. It is difficult to tell whether the cardiac symptoms are due to the fat on the heart or to the general condition of the patient.

The indications for treatment are by a regulated diet and systematic exercise to remove the fat.

2. There is a fatty degeneration of the muscular fibres of the walls of the heart.

The heart remains of its normal size, or becomes somewhat smaller. Its walls are soft and lighter colored. The degeneration may involve the walls of both ventricles, or of one, or of part of one. The degeneration may advance so far at some one point that the wall of the ventricle will be ruptured.

*Causes.*—This change in the heart may be secondary to profound anæmia, to long and wasting diseases, to the severe infectious diseases, and to poisoning by phosphorus. It occurs also as a primary disease, and is then especially common in males between the ages of forty and seventy.

*Symptoms.*—Physical signs: The heart is of normal size, or diminished. The impulse is feeble; the first sound is short and feeble; the action may be regular, irregular, fast, or slow. The systolic murmur at the apex, or at the second left interspace, which occurs without valvular lesions, may be present.

There may be pain, referred to the heart, either continuous or spasmodic; or attacks of angina pectoris.

There may be attacks of syncope, dyspnœa on exertion, or constant dyspnœa. Cerebral symptoms: Vertigo, convulsions, loss of consciousness, or coma may be developed. There may be gradual loss of flesh and strength. Dropsy is not a frequent symptom.

Some of the patients never give any symptoms of their cardiac lesion, and die of some other disease.

Some give no symptoms until they have a fatal attack of angina, of syncope, of coma, or of rupture of the heart.

Some give the characteristic symptoms of the disease for months or years.

*Treatment.*—The best that can be done is the regulation of the diet and mode of life, and the use of cardiac stimulants.

#### RUPTURE OF THE HEART.

*Lesions.*—Ruptures of the heart are most common in the anterior wall of the left ventricle near the apex. They also occur in the wall of the right ventricle, the right auricle, the septum between the ventricles and the papillary muscles. The rupture is small, direct, or indirect. The rupture is due to fatty degeneration of the muscle, or to occlusion of one of the branches of the coronary artery.

*Symptoms.*—The rupture may occur during exertion, or while the patient is perfectly quiet.

If the rupture is direct, death is almost instantaneous. If it is indirect, the patients may live for hours or days. They have sudden pain in the heart, vomiting, dyspnœa, prostration, irregular and feeble heart-action.

#### CHRONIC MYOCARDITIS.

*Definition.*—A chronic productive inflammation of the connective-tissue stroma of the heart, resulting in a production of new connective tissue and degeneration of the muscular fibres.

*Lesions.*—The wall of the left ventricle, the septum between the ventricles, and the papillary muscles are the portions of the heart most frequently involved. The inflammation may be diffuse, involving most of the wall of the left ventricle, or circumscribed, involving only a part of it. The changes are usually most marked at the apex of the left ventricle. The coronary arteries and the arteries in other parts of the body regularly show the lesions of chronic arteritis. The left ventricle may be dilated or hypertrophied. There may be thrombi in the left ventricle. In the portion of the heart-wall affected by the inflammation there are found: a new growth of connective tissue, sometimes an infiltration with pus-cells, and degeneration, or atrophy, of the muscular fibres.

The inflamed portion of the heart-wall may rupture, or may be pouched out so as to form an aneurism.

*Etiology.*—In some cases the myocarditis seems to be secondary to occlusion of the coronary arteries, or to an adherent pericardium. In other cases it is due to the same causes which produce chronic productive inflammations in other parts of the body.

*Symptoms.*—The physical signs consist in the evidences of dilatation or hypertrophy, and the extremely irregular action of the heart.

It is possible for the disease to exist without subjective symptoms, but very often they are present.

There may be the consciousness of irregular heart-action, pain, feelings of constriction, or attacks of true or false angina pectoris. The heart's action is irregular, either too rapid or too slow.

There may be attacks of syncope or of coma, or the mental faculties may be disturbed.

Dyspnœa may be due to the impaired heart-action, to the complicating arteritis, or to attacks of contraction of the arteries.

Venous congestion of the liver, stomach, intestines, and kidneys may be established, with the accompanying loss of nutrition. Dropsy is not common.

The patients may die suddenly.

*Treatment.*—The patients regularly get worse from year to year in spite of treatment. At first the cardiac stimulants may alleviate symptoms, but later in the disease they are of no service and opium is the only drug that gives any relief.

## THE NEUROSES OF THE HEART.

## THE IRREGULAR HEART.

There is a large class of cases of disturbance of the action of the heart without structural disease for which we have no good name, and which we cannot as yet subdivide in a satisfactory manner.

The heart's action may be too rapid, or irregular, or intermittent, or exaggerated, or too slow. These changes in the heart's action either occur in paroxysms, or continue for long periods; they only occur after exciting causes, or continue when no cause can be discovered.

Such an irregular heart-action is accompanied with consciousness of the heart's action; abnormal cardiac sensations; feelings of constriction, displacement, dropping of blood, etc.; more or less mental anxiety; vertigo, headache, fulness of the head, syncope, and sleeplessness; loss of flesh and strength; disturbances of digestion.

Many of the cases are mild and easily relieved, but others give serious symptoms, and are but little benefited by treatment.

The diagnosis of many of the cases is easy; but it may be difficult to distinguish this condition from exophthalmic goitre, chronic myocarditis, disease of the coronary arteries, the fatty heart, or stenosis of one of the valves without a murmur.

The physical signs are the change in the action of the heart, and sometimes a murmur at the second left space, or at the apex.

To the cases in which the heart's action is exaggerated the name of "Palpitation" is often given. It can be seen and felt that the contractions of the ventricles are unnaturally forcible. The mere consciousness of the heart's action is a discomfort, and in addition there may be the feelings of pain or constriction.

The name of "Tachycardia" is given to the cases in which the heart-action is too rapid, whether forcible or feeble. Excluding cases of organic disease of the heart and of exophthalmic goitre, there remains a class of cases in which the rapid heart-action seems to be a neurosis. The tachycardia occurs in paroxysms, or is continuous for long periods. The patients may have some uncomfortable feelings about the heart, but the most noticeable thing about them is their loss of nutrition. Generally



speaking these patients do not improve until the heart beats more slowly.

The names of "Cardiac Asthenia," or "Heart Exhaustion," are given to the cases in which the heart's action is too feeble, although there is no disease of the heart. The patients are often obliged to stay in bed. If they sit up they feel faint, or lose consciousness. The heart's action is feeble, the pulse small and compressible, and generally increased in frequency. There may be uncomfortable feelings about the heart, but no pain. The skin is cold, the appetite poor, the bowels constipated. The patients are sleepless, nervous, and depressed. In most of these cases the cardiac symptoms are only the most prominent symptoms of the condition of neurasthenia.

*Treatment.*—We first try to find out if there is an exciting cause for the disturbed heart-action which can be removed or treated. Such causes are tobacco, coffee, tea, disturbances of digestion, anæmia, and hysteria.

If such an exciting cause can be discovered and be removed, or successfully treated, we may be able to cure the cardiac disturbance; but it may well be that all attempts at treatment on these lines turn out to be useless, and that we have to try to directly influence the action of the heart. For this purpose we employ digitalis, convallaria, caffeine, strophanthus, potassium iodide, aconite, belladonna, or opium, separately, or combined in various ways.

The general management of these patients is a matter of much importance. Some of them need rest in bed, others require graduated exercise. Much can be done by the careful use of hot and cold baths, and of massage. The diet must be so regulated that the patients get enough to eat. All functional disturbances of the stomach and bowels are to be corrected.

#### ANGINA PECTORIS.

A disease characterized by pain, oppression, and a sense of impending death.

It is agreed that the disease is a neurosis, although organic changes of the heart or aorta may be present. It is also recognized that there are two types of the disease, one with contraction of the arteries, and one with irregular heart-action.

*Lesions.*—While in some of the fatal cases of angina pectoris

no lesions of the heart or arteries are found, yet in a still larger number such lesions exist. Inflammation of the arch of the aorta, of the coronary arteries, or of the endocardium; degeneration of the wall of the heart; and changes in the cardiac plexus of nerves have been described.

*Causes.*—The disease is most common in persons over fifty years of age. It is much more frequent in males than in females. The attacks may be brought on by bodily or mental exertion. Concerning the real causation of the disease we are still ignorant.

*Symptoms.*—1. Pain. "Without pain the complaint does not exist." The pain is referred to the precordial part of the lower sternal region, exceptionally to the middle or upper sternal region; with the pain there may be tenderness on pressure. The pain radiates to the mid-dorsal spine, to the left or to both sides of the neck and occiput, to the left shoulder and arm, to both arms, rarely to the right arm alone, to the left leg, or to both arms and the left leg. With the pain there may be feelings of numbness and tingling. The pain is severe and occurs in paroxysms, which last for minutes or hours.

2. There is a feeling of cardiac oppression and a sense of impending death which produces a profound impression on the patient.

3. The pulse varies with the character of the attack, and with the condition of the heart. During the paroxysms it is either a high-tension pulse, or a feeble and irregular pulse.

4. The breathing also varies in the different cases. There may be real dyspnoea with cyanosis, or only a feeling of dyspnoea, or nearly natural breathing.

5. The alimentary canal. There may be violent and continued eructations of gas, or vomiting.

The mind remains clear; there may be slight, or severe general convulsions; the patients may lose consciousness, or they may have vertigo.

The expression of the face is anxious, the skin is cold and often bathed in perspiration.

The attacks usually come on suddenly and without warning. They may, however, be preceded by abnormal cardiac sensations, or be excited by certain muscular movements, or occur at regular intervals.

Each attack may last for a few minutes, or for several hours. A patient may have only a single attack, from which he recovers,

or during which he dies. He may have a number of attacks, any one of which may prove fatal; or after a number of attacks there may be no further recurrence.

The prognosis of the disease is very serious, but yet there are a considerable number of persons who suffer from one or more attacks and entirely recover.

*Treatment.*—If the attacks are accompanied with contraction of the arteries and a pulse of high tension they can often be relieved by inhalations of the nitrite of amyl, subcutaneous injections of morphine, or of nitro-glycerine.

If they are accompanied by a feeble and irregular pulse without contraction of the arteries we employ subcutaneous injections of digitaline and of whiskey.

Between the attacks any existing disease of the heart may require treatment.

#### PSEUDO-ANGINA PECTORIS.

We use this name to designate a somewhat irregular group of cases, which present the common feature of attacks of disturbance of the action of the heart.

*Lesions.*—There may be chronic inflammation of the coronary arteries, fatty degeneration of the heart, inflammation of the arch of the aorta, or no discoverable lesion.

*Symptoms.*—Pain may be absent altogether. When present it is not as severe as the pain of true angina, but is referred to the same regions.

The patients may be much alarmed about themselves, but do not have the feeling of impending death.

The heart's action is feeble and irregular, although sometimes it may seem to be exaggerated.

The breathing may be hurried and labored. There may be vomiting and pain in the stomach.

The attacks come on suddenly, they last for minutes, days, or weeks. In the longer attacks the symptoms are not continuous.

The patient may die in any of the attacks, or after one or more such attacks there may be no further recurrence.

*Treatment.*—During the attacks we use cardiac stimulants; between the attacks we try to improve the general health.

## EXOPHTHALMIC GOITRE.

Basedow's disease. Graves's disease.

The cases are characterized by rapid heart-action, enlargement of the thyroid gland, and protrusion of the eyeballs.

*Lesions.*—After death the thyroid gland is found simply hypertrophied, there is nothing in the orbits, the left ventricle of the heart may be hypertrophied. In a few cases in the ganglia of the cervical sympathetic there have been found degeneration of the nerve-cells and a new growth of connective tissue.

*Causes.*—The disease is much more common in women than in men, and especially during early adult life.

Fright, or severe mental shock, or over-fatigue are believed sometimes to act as causes. Rheumatism occurs as an antecedent condition in a number of cases. The association of the disease with other nervous disorders, such as epilepsy, hysteria, chorea, diabetes, and insanity has often been noticed. In women, anæmia often precedes the disease. There is, in some families, an hereditary predisposition.

*Symptoms.*—The symptoms are usually developed slowly and gradually, but exceptionally are well marked within a few days.

There are three cardinal symptoms: a rapid heart-action, goitre, and exophthalmos. The rapid heart-action is the most constant symptom, the most important, and the one first developed. The heart's action is rapid, regular, forcible, and after a time attended with hypertrophy of the left ventricle.

The enlargement of the thyroid gland follows the rapid heart-action; it is usually, but not always present.

The exophthalmos is the least constant of the three symptoms, and the last to be developed.

Besides the three characteristic symptoms of the disease, there may be others.

There may be tremor affecting the muscles of the whole body, or only those of the arms. It is a tremor like that which is noticed when the muscles are over-fatigued. It is more obvious when the patient is flurried, and sometimes may be noticed only under such conditions. It is more perceptible when the patient is sitting up than when lying down. It only interferes with the more delicate movements of the hands, such as writing or sewing.

There may be painful cramps of some of the muscles, especially at night.

Occasionally there are slight and temporary elevations of temperature.

Sweating is frequent and sometimes excessive.

There may be a brown pigmentation of the skin of the face and of other parts of the body.

Anæmia is very frequently present, especially in young women.

In the severe cases there is well-marked loss of flesh and strength.

Many of the patients become nervous and irritable, hypochondriacal, or hysterical. Some become fairly insane.

The course of the disease is usually protracted for years ; but cases are reported which have lasted only a few months, or days.

Some of the cases recover altogether, some only in part. Death is due to some intercurrent disease, to loss of nutrition, to pressure on the trachea by the thyroid tumor, to syncope, or to attacks of dyspnœa.

*Treatment.*—The indications are: To improve the general health, to cure the anæmia, and to regulate the action of the heart. For the heart-action digitalis, strophanthus, belladonna, or aconite are given, and either the galvanic or faradic current may be applied to the neck.

Good results are reported after removal of the thyroid gland.

## THE ARTERIES.

### CHRONIC ARTERITIS.

*Synonyms.*—Arterio-capillary fibrosis, arterio-sclerosis.

*Definition.*—A chronic productive inflammation of the walls of the arteries, which involves principally their inner and middle coats, and is often accompanied by degeneration.

*Lesions.*—This form of inflammation occurs most frequently in the aortic system of arteries, but it is also seen in the branches of the pulmonary artery. Only a part of the aorta is involved, or the whole of the aorta, or the aorta with most of its branches, or the arteries of some particular part of the body.

There is often chronic productive inflammation of one or more of the viscera.

*Causes.*—It is evident that these morbid changes are caused by lead-poisoning, alcohol, gout, and syphilis; that the disposition to them is hereditary in some families; that they constitute one of the regular senile changes; that they are often associated with chronic diseases of the viscera; that the patients can be unconscious of their existence, and that, on the other hand, they can cause most distressing symptoms, and even death. Chronic arteritis is seen so often in persons over fifty years of age, in fact but few such people have normal arteries, that we are apt to forget that it is also to be seen in adults and children.

Thoma and others believe that the changes in the walls of the arteries are due to slowing of the blood-current. They teach that :

1. Every long-continued slowing of the blood-current causes contraction of the middle coat of the aorta, and, if this is not sufficient to accelerate the blood-current, to a growth of connective tissue in the intima.

2. Primary diffuse and nodular arterio-sclerosis depends upon a weakening of the wall of the blood vessel due to constitutional conditions. This is followed by dilatation of the vessel, slowing of the blood-stream, and then the growth of connective tissue in the intima.

3. Secondary arterio-sclerosis is caused by slowing of the blood-current produced by changes of the circulation in the capillary vessels.

It appears to me that the most practical way to look at all these changes in the arteries is to consider them the results of a combination of chronic productive inflammation and degeneration occurring in connective tissue. We shall then think of the arteries as we do of the endocardium, or the liver, or the kidneys, as a definite part of the body liable to become the seat of chronic inflammation from the same causes as those which produce similar changes in other parts of the body.

*Lesions :* 1. *The Small Arteries.*—*a.* The simplest change in the small arteries is an increase in the size and number of the endothelial cells. This is best seen in the arteries in miliary tubercles and in the small gummata.

- b.* There is a growth of new connective tissue from the endothelium, which encroaches upon the lumen of the artery and finally occludes it. The growth forms a ring on the inside of the intima, thicker in some places than at others.

*c.* There is a thickening of the inner coat beneath the endothelium. The lumen of the artery is irregularly narrowed, while in other places the wall of the artery is thinned. Besides the thickening of the inner coat, the middle and outer coats may also be thickened.

*d.* The whole wall of the artery is thickened and replaced by dense connective tissue.

2. *The Large Arteries.*—In the large arteries altogether the most frequent change is the thickening of the intima. This is often present in arteries which look normal to the naked eye. Besides the thickening of the intima there is often, in addition, a thickening of the middle and outer coats, or a replacement of the muscular coat by connective tissue. When all the coats are thickened in this way, the arteries often become elongated and tortuous. Occasionally there are areas of degeneration in the thickened wall of the artery, or even infiltration with the salts of lime.

3. *The Aorta.*—The changes in the aorta differ from those in the arteries by reason of the combination of degeneration and necrosis with the growth of new tissue due to the chronic inflammation, by the frequency of calcification, and by the liability of the outer coat to purulent infiltration. We find, therefore, in the aorta:

*a.* Simple thickening of the inner coat by new connective tissue.

*b.* Degeneration and softening of the inner and middle coats.

*c.* Calcification of the inner and middle coats.

*d.* Infiltration of the outer and middle coat with pus-cells.

*e.* Thinning and atrophy of the inner and middle coats.

*f.* The formation of thrombi on the roughened surface of the inner coat.

*Symptoms.*—It must be admitted that we find the lesions of chronic arteritis far advanced in persons who have never given symptoms referable to the arteries. It must also be admitted that chronic disease of the lungs, heart, liver, or kidneys is so often associated with chronic arteritis as to obscure the clinical picture. But allowing for all these there remains a large class of cases in which chronic arteritis is the disease.

1. *Chronic Inflammation of the Cerebral Arteries.*—The cerebral symptoms belonging to the inflammation of these arteries have already been described.

2. *Chronic Inflammation of the Medium-sized and Smaller Arteries Throughout the Body.*—At first for a number of years the patients only suffer from impaired nutrition, a disposition to become anæmic, and attacks of dyspnœa. It can be seen and felt that the walls of the temporal and radial arteries are thickened and that the left ventricle of the heart is hypertrophied. At the times when the patient has dyspnœa the tension of the pulse is much increased.

For a considerable length of time the nutrition and the anæmia can be improved by climate and by diet. The attacks of dyspnœa can be controlled by the drugs which dilate the arteries. But sooner or later the patients get worse. Some of them get up a dyspnœa that cannot be controlled, the action of the hypertrophied heart fails, and the patients after suffering for weeks or months with the most distressing dyspnœa die. In other cases death takes place with cerebral symptoms—sudden unconsciousness, aphasia, or hemiplegia. Cases of this character are of ordinary occurrence. Much less common are the cases in which the symptoms run a short and rather acute course. For example, in the “Transactions of the London Pathological Society for 1892” we find the following history :

A girl, aged eleven, in the summer of 1891 had a week's illness, probably to be attributed to infarctions of the lung. In January, 1892, similar symptoms came on, the pain being first in the left side, later in the right ; the cough, with blood-stained sputum, persisted up to the end of the illness. On February 7th the legs became œdematous, and a few days later the face. It was supposed that she was suffering from acute nephritis. The urine contained a little albumin, and was very much reduced in quantity. The child seemed to die of uræmic poisoning. At the autopsy all the arteries of both the aortic and pulmonary systems were found to be diseased. There was consolidation with infarctions of portions of both lungs. Both renal arteries were occluded by thrombi. There were small clots in each of the lateral lobes of the cerebellum. There was hypertrophy and dilatation of the left ventricle of the heart.

3. Chronic inflammation of the femoral arteries and their branches cause gangrene of the toes, or of the legs.

4. Chronic inflammation of the aorta just above the aortic valves may narrow the coronary arteries and cause attacks of angina pectoris.



5. If the whole of the arch of the aorta, with more or less of the rest of the aorta, are involved the patients may have symptoms which resemble those of organic heart disease.

The symptoms come on in attacks, the intervals between the attacks are shorter as the disease advances. The cases vary as to the character of the attacks.

In some patients there is dyspnœa on exertion, or spasmodic dyspnœa. The spasmodic dyspnœa is not attended with any evidence of contraction of the bronchi, or of the arteries.

In others there are attacks of pain like those of true, or of false, angina.

In others there are repeated attacks of epigastric pain and vomiting.

Or there may be congestion and œdema of the lungs, or chronic congestion of the kidneys, or general dropsy.

The nutrition of the patients gradually suffers and they lose flesh and strength. The ventricles of the heart are often dilated, or hypertrophied, or both. There may be a systolic murmur, or a double murmur, and dulness on percussion over the arch of the aorta.

The natural course of the disease is to grow steadily worse and to terminate fatally, but yet for a time the symptoms are not continuous, but come on in attacks.

6. The inflammation of the aorta may be accompanied with the formation of thrombi on its roughened inner surface.

Large thrombi in the arch of the aorta may cause urgent dyspnœa, cyanosis, vomiting, prostration, absence of the heart-sounds and of arterial pulsation, and death within a few days.

Large thrombi in the abdominal aorta produce pain, tenderness, and loss of power in the legs, pain and paralysis of the rectum, and finally gangrene of the legs.

Fragments of such thrombi may be detached and lodged as emboli in the arteries of the brain or of the extremities.

#### ANEURISMS OF THE AORTA.

Fusiform and sacculated aneurisms occur either separately or together. The fusiform dilatations may involve the arch alone or the whole aorta. The sacculated aneurisms have a small or a large opening. The sacs themselves are small, or reach a large size, they are either single or multiple.

*Causes.*—Aneurisms of the aorta are especially common in males between the ages of thirty and fifty years, in persons who have constitutional syphilis; in those who have chronic inflammation of the aorta; and in those whose occupation involves severe muscular exertion.

*Situation.*—Sibson gives the seat of 860 cases of aneurism of the aorta as follows:

Of the arch of the aorta, 480.

At the sinuses of Valsalva, 87.

Of the ascending portion of the arch, 141.

On the transverse portion of the arch, 120.

Of the ascending and transverse portions of the arch together, 112.

Of the descending portion of the arch, 72.

Of the transverse and descending portions of the arch together, 20.

Of the whole arch, 28.

Of the thoracic aorta, 71.

Of the abdominal aorta at the cœliac axis, 131.

Of the lower part of the abdominal aorta, 26.

*Symptoms.*—These are due to the presence of the aneurismal tumor, to its pressure on the surrounding parts, and to its effect on the circulation of the blood. The presence of the tumor is more or less easily detected, according to its size and position.

The large tumors push out the wall of the chest or of the abdomen, can be seen and felt to pulsate, and give a double murmur and a thrill. Smaller tumors within the thorax only give dulness on percussion, with sometimes in addition a double murmur. Very small and deeply situated aneurisms give no physical signs.

Fusiform aneurisms can hardly be said to give a tumor in the thorax. They may give dulness on percussion and a systolic murmur. In the abdomen the dilated artery can be felt, its exaggerated pulsation recognized, and a systolic murmur heard over it.

The aneurisms are for the most part so situated that they press upon the adjacent tissues and so produce a variety of symptoms. Pressure on the descending vena cava causes venous congestion of the head and neck. Pressure on the pulmonary artery, on the right auricle, and on the upper part of the ventricles, interferes with the action of the heart and causes conges-

tion of the lungs. Pressure on the large bronchi and on the trachea sets up inflammation of their walls and mucous membranes. This inflammation often extends up the trachea to the larynx and produces cough and dyspnœa, or down the bronchi into the lungs and sets up a broncho-pneumonia. The pressure also directly produces dyspnœa and tubular breathing which can be heard over the lungs. Pressure on the œsophagus causes difficulty in swallowing. Pressure on the recurrent laryngeal nerve produces paralysis of one of the vocal cords. Pressure on the brachial plexus of nerves produces pain. Pressure on the vertebral column regularly causes erosion of the bones with very great pain.

The aneurisms and the accompanying inflammation of the aorta may cause venous congestion throughout the body, and dilatation and hypertrophy of the left ventricle of the heart.

All sacculated aneurisms are liable to rupture.

#### ANEURISMS AT THE SINUSES OF VALSALVA.

These aneurisms are sacculated, and are most frequently given off from the right coronary sinus. Small aneurisms in this situation give no symptoms until the time comes when they rupture within the pericardium and cause sudden death. Larger aneurisms may compress and erode the pulmonary artery, the right auricle, the right ventricle, or the descending vena cava.

Corresponding to the seat of the aneurism there is an area of dulness or a pulsating tumor, either to the right or left of the sternum, at about the third interspace.

There is a murmur in thirty-four per cent. of the cases.

Dyspnœa in thirty-eight per cent.

Cough in twenty-four per cent.

Pain in twenty-one per cent.

Enlarged heart in twenty-nine per cent.

Rupture of the aneurism within the pericardium in forty-eight per cent.

Into the pulmonary artery in  $13\frac{5}{16}$  per cent.

Into the right auricle in  $8\frac{5}{16}$  per cent.

Into the right ventricle in five per cent.

Into the left ventricle in five per cent.

## ANEURISMS OF THE ASCENDING PORTION OF THE ARCH OF THE AORTA.

These aneurisms are sacculated in fifty-one per cent., fusiform in thirty-seven per cent., of the cases. The ascending portion of the arch of the aorta rests upon the pulmonary artery and the left side of the trachea. On its right side is the superior vena cava; on its left side the pulmonary artery. Most of the sacculated aneurisms are given off from the anterior and right aspects of the wall of the aorta, and a comparatively small number from the posterior and left aspects.

They give an area of dulness, or a pulsating tumor to the right of the sternum, at the second interspace; much less frequently on the left side of the sternum. They may compress the right lung, the vena cava, the left lung, the trachea, the œsophagus, the pulmonary artery, the right bronchus, or the left bronchus.

A murmur is present in twenty-three per cent. of the cases, dyspnœa in fifty-one per cent., cough in thirty-six per cent., stridor in four per cent., pain in the chest in twenty-nine per cent., pain in the neck and shoulders in ten per cent., pain in the right arm in two per cent.

The patients remain in good health and without symptoms up to the time of their death in eleven per cent. of the cases.

Rupture of the aneurism occurs in fifty-seven per cent. of the cases. The rupture is through the wall of the thorax in eight per cent. of the cases, into the pericardial sac in twenty-two per cent., into the pulmonary artery in four per cent., into the right auricle in one per cent., into the descending vena cava in five per cent., into the trachea in one per cent., into the right bronchus in three per cent., into the right lung in five per cent., into the left lung in two per cent., into the right pleura in two per cent., into the left pleura in four per cent.

## ANEURISMS OF THE TRANSVERSE PORTION OF THE ARCH OF THE AORTA.

The transverse portion of the arch passes backward and to the left, over the left bronchus. The left recurrent laryngeal nerve passes behind and against this portion of the arch. The carotid subclavian, and innominate arteries are given off from it.

These aneurisms are most frequently sacculated. The larger number are given off from the posterior wall of the aorta. A smaller number from the anterior or upper wall.

Those given off from the posterior wall soon compress the trachea, or bronchi, or œsophagus, and the recurrent laryngeal nerve. The patients very early have dyspnœa, cough, stridor, paralysis of one of the vocal cords, tubular breathing over one or both lungs, but the physical signs of the tumor are absent or obscure until it reaches a large size. These aneurisms are apt to rupture into a bronchus, into the trachea, or into the œsophagus.

The aneurisms given off from the anterior and upper wall soon give the physical signs of a tumor. They partly occlude the large arteries given off from this part of the aorta, and diminish the pulsation of the carotid and radial arteries. They must, however, attain a considerable size before they can exert much pressure on surrounding parts.

A pulsating tumor at or above the level of the manubrium is found beneath the sternum in nineteen per cent. of the cases; to the right of the sternum in  $12\frac{5}{10}$  per cent.; to the left of the sternum in  $12\frac{5}{10}$  per cent.

A murmur is present in fifteen per cent. of the cases, dyspnœa in seventy-one per cent., cough in fifty-seven per cent., hæmoptysis in nineteen per cent., stridor in  $47\frac{5}{10}$  per cent., dysphagia in thirty-one per cent., pain in thirty-six per cent., weakening of one radial pulse in twenty-six per cent.

The aneurisms rupture externally in six per cent. of the cases; into the pericardium in two per cent., into the right pleura in two per cent., into the left bronchus in four per cent., into the left pleura in five per cent., into the œsophagus in six per cent., into the pulmonary artery in one per cent., into the descending vena cava in one per cent., into the trachea in ten per cent.

#### ANEURISMS OF BOTH THE ASCENDING AND TRANSVERSE PORTIONS OF THE ARCH OF THE AORTA.

These aneurisms are fusiform in eighty-five per cent. of the cases. Most of them soon reach a considerable size. There is dulness on percussion, or a pulsating tumor, most frequently on the right side of the sternum, often beneath the sternum, less often on the left side of the sternum.

The pressure of the tumor presses forward and erodes the sternum and the ribs. The trachea is compressed in thirty-three per cent. of the cases, the superior vena cava in ten per cent., the œsophagus in nine per cent.

Dyspnœa is present in seventy-four per cent. of the cases, cough in forty-seven per cent., hæmoptysis in ten per cent., stridor in seventeen per cent., dysphagia in twenty-one per cent., pain in the chest in thirty per cent., pain in the back in ten per cent., pain in the arms in eighteen per cent., pain in the neck in four per cent.

The right pulse is feeble in six per cent. of the cases, the left pulse in three per cent.

The aneurisms rupture into the pericardium in ten per cent. of the cases, into the pulmonary artery in  $1\frac{5}{10}$  per cent., into the vena cava in four per cent., into the trachea in four per cent., into the right pleura in three per cent., into the left pleura in one per cent.

#### ANEURISMS OF THE DESCENDING PORTION OF THE ARCH OF THE AORTA.

The descending portion of the arch lies at first on the left lung and the œsophagus, and at last rests on the left half of the body of the fifth or sixth dorsal vertebra, where the œsophagus is on the right side of the aorta. In front of it are the left bronchus and the pulmonary artery.

The aneurisms are sacculated in eighty-one per cent. of the cases, fusiform in eight per cent.

The aneurisms are given off from the posterior and left walls of the aorta.

The vertebræ are eroded in forty-two per cent. of the cases, the left ribs in front in four per cent., the trachea is compressed in  $12\frac{5}{10}$  per cent. of the cases, the left bronchus in  $37\frac{5}{10}$  per cent., the œsophagus in thirty-one per cent., the left lung in forty-eight per cent., the right lung in six per cent.

The presence of the aneurismal tumor cannot usually be made out by physical signs until it has reached a considerable size.

A murmur is present in twenty-three per cent. of the cases, dyspnœa in fifty per cent., cough in forty-six per cent., stridor in

twenty-five per cent., dysphagia in thirty-three per cent., pain in the chest in fifty-two per cent.

The aneurisms rupture into the left pleura in twenty-three per cent. of the cases, into the right lung in six per cent., into the right pleura in twelve per cent., into the trachea in four per cent., into the left lung in four per cent.

#### ANEURISMS OF THE THORACIC AORTA.

These are sacculated in thirty-five per cent. of the cases, fusiform in twenty-eight per cent.

Erosion of the vertebræ occurs in seventy-four per cent. of the cases. The œsophagus, the left lung, or the right lung may be compressed, the heart may be displaced forward, there may be a pulsating tumor in the dorsal or axillary regions.

A murmur is present in four per cent. of the cases, dyspnœa in twenty-six per cent., cough in twenty-four per cent., dysphagia in nine per cent., pain in the back in forty-three per cent., in the chest in twenty-nine per cent., in the abdomen in thirteen per cent., in the left lumbar region in nineteen per cent.

The aneurisms rupture into the trachea in two per cent. of the cases, into the left lung in thirteen per cent., into the left pleura in twenty-six per cent., into the right lung in six per cent., into the right pleura in thirteen per cent.

#### ANEURISMS OF THE ABDOMINAL AORTA.

These are usually sacculated. The larger number are situated near the cœliac axis. They are given off with about equal frequency from the anterior and posterior walls of the aorta.

Those on the posterior walls very early erode the vertebræ. They give pain referred to the vertebræ and radiating down. While the tumors remain of small size it is often difficult to make them out.

The anterior aneurisms give pain referred to the epigastrium and the anterior portion of the abdomen. The tumors are easily made out by palpation.

These aneurisms rupture into the peritoneal cavity, behind the peritoneum, into the mesentery, and into the right or left pleural cavity. The blood may escape at once into the perito-

neal cavity and cause death, or it may be for a time held in behind the peritoneum and a false aneurism be thus formed.

There may be incomplete or complete obstruction of the aorta by thrombi extending from the aneurismal sac.

*Treatment.*—The medical treatment of aneurism of the aorta consists in determining for each patient, experimentally, whether he is better for rest or for regulated exercise, and in the administration of the iodide of potash. The iodide is given in doses of from five to twenty grains, three or four times a day. In favorable cases the aneurism is diminished in size, the symptoms are alleviated, and life is prolonged.



## THE ŒSOPHAGUS.

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### STENOSIS.

1. Congenital stenosis of the œsophagus is rare. It is most frequently seen in children, who die early. Exceptionally, the patients survive to adult life, but with constantly increasing difficulty in swallowing.

2. Stenosis by compression is due to the pressure of aneurisms, of tumors of the thyroid, and of tumors of the neck and the thorax. The difficulty in swallowing is only one of the symptoms of the tumor.

3. Stenosis from tumors of the posterior wall of the pharynx, either polypoid or diffuse.

4. Cicatricial stenosis from the contraction following the lesions inflicted by irritant poisons. These require surgical treatment.

5. Stenosis due to syphilitic inflammation of the wall of the œsophagus.

6. Carcinoma of the œsophagus.

The new-growth, of flat-celled epithelial type, begins at some part of the wall of the œsophagus, and soon surrounds it. There are masses of new-growth, and deep ulcers; either may be the predominant feature. The new-growth involves only one or two inches of the œsophagus, or a much larger portion. The lower half of the œsophagus is the portion most frequently involved. The growth may remain confined to the wall of the œsophagus, or may extend to the trachea, bronchi, lungs, pleura, pericardium, and vertebræ. Metastatic tumors may be formed in the lymphatic glands, lungs, and liver. A cancer of the stomach may be developed at the same time with a cancer of the œsophagus.

The disease occurs most frequently in males over forty years of age.

The first symptom is difficulty in swallowing. This begins with difficulty in swallowing large pieces of solid food, then smaller pieces cannot be swallowed, and, finally, not even liquids. The progress of the dysphagia is gradual, and often intermittent.

The next symptom is pain, referred to the upper end of the sternum.

It is not until the tumor has existed for some time, and the difficulty in swallowing is considerable, that there is much change in the nutrition of the patients. But after they have once begun to emaciate they get worse very rapidly.

Life may be prolonged for a few weeks by passing tubes through the œsophagus into the stomach, and by the use of rectal alimentation.

7. Spasmodic stricture occurs in young and old persons, in both males and females. There is sometimes a history of hysteria, sometimes one of sudden choking.

The one symptom is difficulty in swallowing, sometimes attended with pain.

The dysphagia may be complete, or partial. It occurs in attacks which last for minutes, days, weeks, or months, and which are repeated at short or long intervals. It differs from the dysphagia of organic disease in that it does not gradually get worse, but is as bad at first as later, and there is as much difficulty in swallowing fluids as solids. The contraction of the œsophagus is just above the stomach. I have seen one case, of ten years' duration, in which there was a dilatation of the œsophagus above the stricture, in which the food collected.

The treatment of spasmodic stricture is fairly satisfactory, but it has to be continued for a long time. It consists in passing a tube of moderate size into the stomach one or more times every day, washing out the stomach, and then introducing fluid food through the tube.

#### DILATATION.

1. Fusiform dilatations of the œsophagus are secondary to stenosis, or occur without discoverable cause. They involve a part, or the entire length, of the œsophagus. The wall of the dilated œsophagus is often thickened.

The symptoms are difficulty in swallowing, vomiting, regurgitation of food, and rumination.

The treatment consists in the dilatation of the stenosis and the feeding the patients with the stomach-tube.

2. Sacculated dilatations are of two kinds.

(a) Small sacs are formed in the lower part of the posterior wall of the pharynx, which afterward become larger.

They are more common in males than in females, and belong to adult life.

How it is that these sacs begin to be formed is not well understood. But as soon as they have reached such a size that the food can enter and remain in them, they rapidly enlarge behind the œsophagus and compress it.

There is difficulty in swallowing, at first slight, but increasing with the pressure of the sac on the œsophagus.

There is regurgitation of the food from the sac, rumination, and vomiting. There may be a tumor in the neck which can be emptied by pressure.

The only treatment which has thus far been employed is to feed the patients with the stomach-tube.

(b) Small sacs are given off from the anterior wall of the œsophagus, at about its middle. They seem to be due to an inflammation of the bronchial glands, an extension of this inflammation to the wall of the œsophagus, and traction.

These sacs give no œsophageal symptoms, but they may perforate and so give rise to inflammatory processes in the mediastinum.

## THE STOMACH.

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The œsophageal end of the stomach is on a level with the eighth dorsal vertebra behind and the sixth left interspace close to the sternum in front. The pyloric end of the stomach is at the level of the eighth costal cartilage, just to the right of the sternum, and is covered by the liver. The œsophageal end of the stomach is fixed, but its pyloric end is movable and often displaced. The greater curvature of the stomach is close to the colon, and its fundus touches the spleen.

The outer surface of the stomach is covered with a connective-tissue membrane—the peritoneal coat; beneath this is the muscular coat; then comes the connective-tissue coat; then a thin layer of muscular fibres, the *muscularis mucosæ*; and lastly, the glandular coat. The glandular coat of the stomach is composed of the peptic tubules which form the gastric juice, and the mucous tubules which form mucus. The mucous tubules are not nearly as numerous as the peptic tubules, and are situated at the pyloric end of the stomach. All the tubules are held together by a stroma of connective tissue. About the blind ends of the tubules are small lymph-nodes. The entire wall of the stomach is well supplied with blood-vessels, lymphatics, and nerves.

It is the function of the stomach to receive the food, to digest certain portions of it, and, after a proper lapse of time, to expel it all, digested and undigested, into the small intestines.

The nitrogenized substances—albumin, gluten, fibrine, caseine, and gelatine—are acted upon by the gastric juice and converted into peptones. The starches are partly changed into sugar, and the fats are softened.

The more thoroughly the process of the stomach digestion is studied, the more evident it becomes that it is a complex one carried out by a variety of ferments. The knowledge of the

subject obtained by the physiological chemists has not yet been put in a shape available to the physician.

In a healthy person, while the food is being received into the stomach and while gastric digestion is going on, the pylorus is closed, but the muscular coat of the stomach is in activity, moving about the contained food. After about four hours the necessary changes in the stomach should be completed, the contents of the stomach expelled through the pylorus, and the organ then remains empty and contracted until the next ingestion of food.

*Methods of Examination.*—The portions of the stomach which are not covered by the wall of the chest or the liver, are available for palpation and percussion. These portions of the stomach are made more evident by causing the patient to take a deep breath and depress the diaphragm.

Tumors and thickenings of the wall of the stomach can usually be felt without difficulty.

If the stomach is dilated, its outlines can sometimes be seen through the anterior abdominal wall, especially when the patient takes deep inspirations. It is not easy to feel the wall of the stomach unless it is thickened, but in some cases the greater curvature can be made out by percussion. It has been recommended to distend the stomach with carbonic dioxide by giving the patient first some bicarbonate of sodium and then tartaric acid, or by pumping air into it, or by filling it with water in order to determine whether the organ is dilated. As a rule, these procedures are to be avoided as unnecessary and not devoid of danger.

Dilatation of the stomach is most easily made out by means of the stomach-tube. The length of tube which has to be inserted before the water will run in and out from the stomach, and the quantity and character of the contents of the stomach give the necessary information.

In most stomachs within an hour after eating a splashing sound can be produced by palpation. This sound is more evident if the stomach is dilated. It is not, however, by itself an evidence of dilatation of the stomach. Similar splashing sounds can be produced in the colon.

The contents of the stomach can be removed through the stomach-tube, by expression or aspiration, and subjected to chemical analysis. It is customary to test for the presence and quantity of HCl, of lactic acid, and of the fatty acids. Of much

more practical consequence is the determination with the tube of the capacity of the stomach for digesting different kinds of food.

In the management of diseases of the stomach the tube is necessarily in daily use. It is important, therefore, that the tube should be of such a size and consistence as to be comfortable to the throat, and that the physician should acquire the dexterity necessary to pass it quickly and easily.

#### DISTURBANCES OF THE FUNCTIONS OF THE STOMACH WITHOUT ORGANIC DISEASE. GASTRIC DYSPEPSIA.

Disturbances of the functions of the stomach without structural disease of its walls are produced in a variety of ways.

1. The habitual ingestion of food in excessive quantities, or of improper quality, may after a time cause gastric symptoms. It must, however, be remembered that gastric symptoms are often due to entirely different causes, and that too little food is as bad as too much.

One of the simplest disturbances due to an excessive ingestion of food is seen in children. A child at some particular meal overfills its stomach. The pylorus remains closed and no gastric digestion takes place. After some hours the child has abdominal pain, rapid heart action, a cold skin, and sometimes marked prostration. Finally it vomits the food in nearly the same condition as when it was taken into the stomach, and is at once relieved of all symptoms.

The same thing can be seen in adults. A large meal is eaten, little or no gastric digestion takes place, the pylorus remains closed, the stomach is distended with undigested food. After a number of hours the patient has the feeling of vertigo or of faintness, or he may fall to the ground unconscious. Then there is a profuse vomiting of the contents of the stomach.

In adults habitual over-eating may cause chronic gastritis and dilatation of the stomach, but it is much more apt to cause symptoms referable to disturbances of the functions of the liver—feelings of lassitude and depression, headache, constipation, diarrhœa, flatulence, an excess of urates in the urine.

The habitual eating of improper food results in an impairment of nutrition, for not only is the improper food bad in itself, but it takes the place of wholesome food.

The treatment of these cases consists in a regulation of the

diet. In the bad cases the patients must be put for a time on the exclusive use of milk, or of scraped beef, but neither of these exclusive diets can be continued with advantage for any great length of time. The use of all sorts of health-foods, peptonized foods, pepsin and pancreatine, is to be avoided. The patients must be educated to eat meat, starches, fats, vegetables, and fruits in proper quantities. In doing this the use of the stomach-tube, about four hours after one of the meals, is of much service as a guide to the particular articles of food which are best digested and remain the shortest time in the stomach.

2. Changes in the gastric juice. The two most marked changes in the gastric juices are : such a change in its composition as renders it unable to perform stomach digestion, or increased quantity and hyperacidity.

(a) Changes in the composition of the gastric juice of such a character that stomach digestion is imperfectly performed.

This condition is seen in persons who have had chronic gastritis for a long time, with changes in the glandular coat of the stomach. It is also seen in young persons in whom there is no evidence of disease of the glandular coat.

The patients have no appetite, or even a distaste for food. In some there is occasional nausea, vomiting, and pain after eating. The bowels are usually constipated. The nutrition suffers noticeably, the patients lose flesh and strength. Besides the gastric symptoms there are often a variety of accessory symptoms of nervous and hysterical character. It is not uncommon to find persons belonging to this class who do not complain of any gastric symptoms, but only of the loss of nutrition and some disturbance of sensation.

On the other hand we must not confound with this class the persons who complain of gastric pain and oppression, loss of appetite and nausea, but in whom gastric digestion is well performed.

The diagnosis is made with the help of the stomach-tube.

There will be found a considerable number of persons in whom more or less partly digested food is always present in the stomach. The problem in these persons is to improve gastric digestion. It would seem as if this could be done by the use of pepsin, or hydrochloric acid, or partly digested foods, but it will be found that no permanent improvement can be effected in this way. There is no one rule for the management of all these

patients, but the general plan is to improve the general health by massage, baths, exercise, and travel, and to feed the patients as well as can be done. The method of feeding has to be found out for each person with the stomach-tube. We try the simple articles of food — milk, eggs, kumyss, meat, beef-juice, gruels, etc.—and see which ones leave the cleanest stomach in the shortest time. In this way we get a guide as to the best kind of food and the proper intervals between taking food. As the patients improve we gradually add other kinds of food. It requires a number of months to get these persons back to a natural condition of health.

(b) The gastric juice may contain an excessive quantity of hydrochloric acid, and be itself produced in too large quantities. The most marked symptoms of this condition are epigastric pains and vomiting. To determine this condition accurately the stomach should be washed out in the evening and its contents aspirated the next morning before the ingestion of food or fluid.

The milder cases are benefited by the use of alkalies, but the only satisfactory treatment is the systematic washing out of the stomach.

Temporary insufficiency of the gastric juice may be caused by a variety of nervous and mental conditions.

3. The muscular coat of the stomach may cease to perform properly its function of moving and expelling the food. This condition is apt to be slowly developed, so that it is not until after several years that it becomes a serious evil. The insufficient action of the muscular coat of the stomach allows food to remain in the stomach from one meal to another, so that the organ is never empty. This constant presence of food may eventually cause chronic gastritis or dilatation of the stomach. The symptoms at first come on in attacks which last for several days; between the attacks the patient is well. As time goes on the attacks become more frequent and of longer duration, and finally the symptoms are continuous. The principal symptom is pain of greater or less severity referred to the region of the stomach. To the pain are regularly added after a time constipation, nausea, vomiting, and finally loss of flesh and strength.

During the earlier periods of their trouble the patients improve under an absolute diet of milk or of meat, but the only really satisfactory treatment is the washing out of the stomach, with a regulated diet.



4. Disorders of the colon—constipation, chronic inflammation, or carcinoma—may be accompanied by gastric symptoms—pain, nausea, and vomiting. In elderly persons, whenever nausea and vomiting are present it is always necessary to think of an accumulation of fæces in the colon as the probable cause.

5. Diseases of the uterus, ovaries, and tubes are rather frequently accompanied by loss of appetite, nausea, and vomiting. These symptoms are continuous, or come on in attacks. There may really be chronic gastritis, or inefficient gastric juice, or retention of food in the stomach in addition. But it will be found that the treatment of the stomach is unsatisfactory unless the disease of the uterus, ovaries, or tubes, can first be cured.

6. In the simple anæmia of young women gastric symptoms are usually present, and in some of the patients are especially severe. Loss of appetite, nausea, and vomiting are often present, hæmatemesis is not rare. The gastric symptoms may be so marked as to make it easy to overlook the anæmia, but this error is readily avoided by the examination of the blood.

In these anæmic patients the gastric symptoms promptly disappear with the cure of the anæmia.

7. Hysterical patients are capable of developing a great variety of gastric symptoms—loss of appetite, perverted appetite, distaste for food, nausea, vomiting, pain, and flatulence.

As a rule these patients are best treated if we disregard the gastric symptoms and employ the regular management for hysterical cases.

8. The habitual use of tobacco and of alcohol will, in some persons, even before chronic gastritis is produced, cause loss of appetite, nausea, and vomiting.

9. Neurasthenia. Of the patients who suffer from the condition of neurasthenia a considerable number have decided gastric symptoms. Generally speaking it is better not to pay too much attention to the stomach, but to rely on baths, massage, exercise, climate, etc. I see neurasthenics, however, who are entirely relieved by lavage of the stomach. This seems to be a simple faithful cure.

#### GASTRALGIA.

Pain in the stomach accompanies an excessive production of hyperacid gastric juice, regurgitation of bile into the stomach, insufficient action of the muscular coat, diseases of the colon, anæ-

mia, hysteria, acute gastritis, chronic gastritis, ulcer of the stomach and cancer of the stomach, and locomotor ataxia. It is closely simulated by the pain of intestinal colic, by the pain due to calculi in the gall-bladder and bile-ducts, and by ulcers of the duodenum.

In some cases of movable kidney the patients complain of gastric pain, nausea, and vomiting.

Whether, in addition, gastralgia occurs as a pure neurosis, it is difficult to say. Probably with greater accuracy in diagnosis the apparent number of such cases diminishes. In those cases in which we do have to make the diagnosis of gastralgia as an independent condition, the drugs which seem to be of the most service are arsenic or quinine.

#### ACUTE DILATATION OF THE STOMACH.

This is a very rare condition. I copy the account of it from Quain's "Dictionary of Medicine."

The earliest case on record is that of a lady mentioned in the fourth volume of the "Pathological Transactions," by Dr. Miller and Dr. Humby. She had been under treatment for piles shortly before her illness, and the abdomen had been observed to have increased in size. She was attacked with vomiting of immense quantities of fluid. The vomiting ceased four days afterward, and the abdomen was found to be greatly enlarged. After death the cause of the abdominal distention proved to be the stomach, which was so much dilated that it was capable of holding ten pints of liquid. Dr. H. Bennett, of Edinburgh, relates a similar case, and attributes the dilatation to a large quantity of effervescing liquid the patient had swallowed to allay his thirst. Dr. Hilton Fagge, in the "Guy's Hospital Reports" (vol. xviii., Third Series), describes two cases that had fallen under his notice, and also mentions that two similar cases had been observed at Guy's Hospital during fourteen years.

*Diagnosis.*—The signs of the dilatation, according to Dr. Fagge, are: 1. A rapidly increasing distention of the abdomen, which is unsymmetrical, the left hypochondrium being full, while the right hypochondrium is comparatively flattened. 2. The existence of a surface-marking descending obliquely toward the umbilicus from the left hypochondrium, and corresponding with the dragged-down lesser curvature of the stomach, this line ap-

pearing to descend with each inspiration. 3. The presence of fluctuation in the lower part of the abdomen. 4. The occurrence of splashing when the distended part of the abdomen is manipulated. 5. The presence of a uniformly tympanitic note over a large part of the distended region when the patient lies flat on his back. Above the pubes, on the other hand, there may be dulness on percussion simulating that of a distended bladder.

The treatment is to empty the stomach by the stomach-tube and feed the patient with nutrient enemata.

#### CHRONIC DILATATION OF THE STOMACH.

For clinical purposes it is convenient to describe three forms of dilatation of the stomach.

1. There is a form of moderate dilatation of the stomach, without stenosis of the pylorus, without chronic gastritis, without failure of stomach digestion. If not relieved by treatment the condition continues for years, although there are often periods of improvement. The two prominent symptoms are pain in the upper part of the abdominal cavity and loss of nutrition. The pain comes on at intervals, which become shorter and shorter until there is pain every day. The loss of flesh and strength are progressive until the patients can hardly walk.

With these two cardinal symptoms are often associated a variety of accessory symptoms. Some patients complain of dyspnœa, apparently diaphragmatic in character. In some the heart's action is increased in frequency. In women there may be a variety of nervous and hysterical symptoms. The loss of nutrition seems to depend partly on insufficient food, for the patients are apt to leave off one article of food after another with the idea of preventing the pain.

Temporary relief may be obtained by travelling, by an exclusive diet of milk or of meat, by pepsine, bismuth, nux vomica, etc. But the symptoms soon return and grow worse. The only curative treatment is the daily lavage of the stomach. This is very effectual, even in patients who have suffered for a number of years.

2. There is a form of dilatation of the stomach without stenosis of the pylorus, but with chronic gastritis. In these cases the stomach is larger and articles of food are retained in it for a longer time, sometimes for several days. There are pains

in the upper part of the abdominal cavity and a loss of flesh and strength which is carried very far. Nausea and vomiting are regularly developed after a time. When the stomach is first washed out, even after twelve hours' abstinence from food, it will be found to contain a large quantity of undigested material.

These patients get well with lavage, but it has to be kept up for a long time.

3. There is a form of dilatation with stenosis of the pylorus. The stenosis is caused by carcinoma, by ulcers, and by inflammatory thickenings. The dilatations produced in this way are very large and much food is retained. The patients have pain, loss of nutrition, nausea, and vomiting.

The patients are made more comfortable and life is prolonged by lavage, but after a time they do badly, lose flesh and strength and die.

The surgical procedures usually adopted for the relief of the stenosis are : resection of the pylorus, dilatation of the pylorus, and intestinal anastomosis.

#### ACUTE CATARRHAL GASTRITIS.

*Lesions.*—The changes are the same as in all acute catarrhal inflammations—congestion, swelling, at first dryness, later an increased production of mucus, exudation of serum, emigration of white blood-cells. The inflammation may involve the whole of the glandular coat of the stomach, or only its pyloric portion ; it may extend to the duodenum.

*Causes.*—The disease occurs at all ages and in both sexes. It may occur at any time of the year, but is especially common in summer. It is apparently sometimes caused by atmospheric conditions. It frequently complicates the exanthemata, typhoid fever, and epidemic influenza. It may be produced by unwholesome and irritating food. It is of frequent occurrence in persons who have fatty degeneration of the liver. It occurs very often as a primary inflammation. Some persons show a marked predisposition to the disease, and suffer from repeated attacks. This is especially the case in some young children. They will have one or more attacks of acute gastritis every year for several years. As they grow older the attacks are less frequent and less severe.

*Symptoms.*—The symptoms of an acute gastritis may come on

suddenly or they may be preceded by symptoms of gastric indigestion, of subacute gastritis, or by a diminution of the quantity of bile in the stools.

Pain is often complained of. It may be a severe pain, or only a feeling of soreness or of discomfort.

Vomiting is regularly present. It is in proportion to the severity of the gastritis, and is most troublesome during the first days of the inflammation. The patients vomit whatever they take into the stomach, and more or less mucus and serum. In severe cases large numbers of pus-cells and some red blood-cells are found in the vomited mucus and serum. The irritability of the stomach may be so great that neither food nor medicine can be retained.

A rise of temperature accompanies the first days of the inflammation in some patients, but in many there is not at any time a febrile movement. As a rule, the bowels are constipated. But in some patients an acute colitis is developed at the same time as the gastritis, and then there is diarrhœa. There is a good deal of variety as to the length and severity of the attacks, the frequency of the vomiting, the height of the temperature, and the degree of the prostration. Attacks of acute gastritis are not infrequently followed by subacute and chronic gastritis.

*Course of the Disease.*—(1) In infants and young children the vomiting is incessant, and excited by everything which is taken into the stomach; the rise of temperature is often well marked, and the prostration is considerable. The gastritis may be accompanied with or followed by diarrhœa. The symptoms may last only for a few days, or for several weeks. Such a gastritis may not at any time be serious, or it may produce a prostration which lasts for weeks, or it may be fatal.

(2) Mild cases in adults. The patients are not at any time very sick, nor is there a rise of temperature. There are loss of appetite, nausea and vomiting, with a feeling of discomfort or of actual pain referred to the stomach. The tongue is coated, the bowels may be constipated. The attack usually runs its course within a week and terminates in recovery, but the inflammation may pass into the subacute or chronic condition.

(3) Severe gastritis in adults is attended with the same symptoms, but they are more marked.

The patients are sick enough to stay in bed, in some the prostration and feeble heart action are alarming, in some the

loss of flesh and strength are extreme. A febrile movement may be present or absent. The feeling of nausea is constant, the vomiting frequent, the pain and discomfort most distressing. Even a severe attack may run its course within a week, but the attacks often last much longer, and may be succeeded by sub-acute or chronic gastritis.

(4) The inflammation involves either the whole stomach, or the pyloric end of the stomach, the duodenum, and the common bile-duct. The patients then have the symptoms described under the name of simple jaundice.

(5) The inflammation is confined to the pyloric end of the stomach and the duodenum. The symptoms are the same as those of an attack of simple jaundice, but without the jaundice. The patients suffer from general malaise, headache, dulness, drowsiness, vertigo. The tongue is coated, loss of appetite, nausea, occasional vomiting, pain in the epigastric region and sometimes fever are present. The bowels are constipated and the *fæces* light colored.

*Treatment.*—There can be no doubt that in some persons acute gastritis is caused by hot weather. It is important for such persons to pass their summers in a cool climate.

In infants gastritis is often caused by the food. Not that the food is irritating but that it contains pathogenic bacteria and the products of their growth. This is an additional reason for the most scrupulous cleanliness in preparing and sterilizing the food of children.

In any case of acute gastritis it is well to keep the patient in bed. For the first twenty-four or forty-eight hours the irritability of the stomach may be so great that it will retain nothing. When this is the case it is wise to desist altogether from giving food or medicine by the mouth. After this the food is given at first by the teaspoonful and then the quantity gradually increased. The best foods are usually cream and water, koumyss, milk, beef-tea, beef-juice, and scraped beef. Rectal enemata help but very little in nourishing these patients.

The external application of heat over the upper portion of the abdomen by fomentations, hot-water bags, or poultices is grateful to the patient.

Of drugs the sulphate of morphia and codeia are the most valuable. They are to be given in small doses,  $\frac{1}{16}$  to  $\frac{1}{8}$  grain of morphia,  $\frac{1}{12}$  to 1 grain of codeia, at intervals of from one to three

hours. They are to be used in the form of tablets, which dissolve in the mouth, or given hypodermically.

The hydrochlorate of cocaine is sometimes of use for adults, one-tenth grain in tablet form at intervals of from one to four hours.

Ipecac in tablets of one-tenth grain given every hour may exert a specific effect on the gastritis. The oxalate of cerium and bicarbonate of soda are useful drugs, they are best given in milk or in cream and water. A good formula is to mix together in a half-pint measure equal parts of cream, milk, and water, add to this ten grains of oxalate of cerium and twenty grains of bicarbonate of soda; give a teaspoonful every half hour.

When the gastritis involves the pyloric end of the stomach, rectal enemata of very hot or very cold water, and purgation by calomel may be of much service.

#### CHRONIC CATARRHAL GASTRITIS.

It is customary to include several different lesions of the stomach under this name.

*Lesions.*—1. The only evident change in the stomach is an increased production of mucus. This can be washed out of the stomach during life, it is found adherent to the wall of the stomach after death.

2. Besides the increased production of mucus there is also degeneration of the cells of the peptic glands.

3. There is a growth of connective tissue between the gastric tubules, with deformity and atrophy of the tubules. In some cases the new connective tissue forms little polypoid tumors which project inward.

4. With cardiac disease and with cirrhosis of the liver there may be a well-marked chronic congestion of the mucous membrane.

5. There may be hypertrophy of the muscular and connective coats at the pylorus producing stenosis, and with this there is often dilatation of the stomach.

6. There may be a more diffuse hypertrophy of the muscular and connective-tissue coats with marked diminution in the size of the stomach.

7. The stomach may be considerably dilated without stenosis of the pylorus.

When we say that a patient has chronic catarrhal gastritis we mean that either :

The mucous glands are constantly producing too much mucus.

Or that in addition the functions of the peptic glands are disturbed.

Or that the structure of the glandular coat is so impaired by disease that gastric digestion is seriously interfered with.

Or that the mucous membrane is constantly in the condition of chronic congestion.

Or that from stenosis of the pylorus or relaxation of the muscular coat, food is retained in the stomach.

Or that the stomach is dilated or contracted.

*Etiology.*—A considerable number of cases of chronic gastritis follow acute or subacute gastritis. This is especially true of the gastritis which complicates epidemic influenza, typhoid fever, and so many of the infectious diseases.

Cardiac disease, pulmonary emphysema, and cirrhosis of the liver produce, first, chronic congestion and then chronic gastritis.

Pulmonary phthisis, chronic nephritis, and gout are often accompanied by chronic gastritis.

The habitual use of improper food, of alcohol, and of drugs are ordinary causes.

The same climatic conditions which predispose to chronic naso-pharyngeal catarrh and to chronic bronchitis have the like effect as regards chronic gastritis.

*Symptoms.*—First among the symptoms comes pain, varying from a mere feeling of oppression and discomfort to the most severe and agonizing pain. The pain is in some cases due to the presence of food in the stomach. It comes on at first only with a considerable quantity of food, but later even small fragments of solid food or spoonfuls of liquid food will excite it. Very often patients learn to vomit of their own accord in order to relieve the pain.

In some persons the pain seems to accompany gastric digestion and to cease when this is finished.

In some cases the pain seems to depend on hyperacidity and hypersecretion of the gastric juice.

With alcoholic gastritis there may be a peculiar feeling of soreness in the stomach, which is commonly called heartburn.

Then there are many cases in which we can only say that so



long as the patients have gastritis they have pain, and as the gastritis improves so does the pain.

As a rule the pain is at first not constant, but comes on in attacks, which become more frequent and of longer duration as the gastritis progresses.

It is often difficult to distinguish the pain of chronic gastritis from that belonging to ulcer of the stomach, ulcer of the duodenum, biliary calculi, intestinal colic, movable kidney, disease of the uterus, tubes, and ovaries, anæmia, hysteria, and neurasthenia.

Nausea and vomiting are also regular symptoms. The nausea belongs to the early morning hours, but may persist throughout the day. It may show itself as a disgust for food, or as a feeling of anxiety and depression of spirits, or as the actual sensation of impending vomiting.

The vomiting is most frequently a vomiting of food, either in considerable quantities, or vomiting kept up so long as the smallest particle of food is left in the stomach. In some cases of alcoholic gastritis the stomach empties itself about an hour after nearly every meal.

In other cases large quantities of brownish fluid, mixed with mucus and food, accumulate in the stomach and are vomited from time to time. This is especially the case when the stomach is dilated. Regurgitation of an acid fluid before breakfast in the morning is especially common with alcoholic gastritis. In a few cases the patients will vomit nearly every day as much as half a pint of pure mucus.

There are rare cases in which vomiting is kept up as a habit long after the real reasons for it have stopped.

Vomiting of blood may occur in the course of any chronic gastritis, but is much more likely to occur if cirrhosis of the liver, or fatty liver, exist at the same time. Usually the hemorrhages are small, but with cirrhosis of the liver large quantities of blood are coughed up.

The vomiting may occur in attacks with intervals of days or weeks, or it comes every day. I have known a man to vomit every day for twenty years.

Vomiting is also caused by diseases of the uterus, tubes, and ovaries, by an accumulation of fæces in the colon, by anæmia and by hysteria.

Retention of food in the stomach is especially a feature when there is stenosis of the pylorus or dilatation of the stomach, but

it is also found with chronic gastritis. A considerable retention of food is regularly attended with vomiting.

Constipation is often present. It seems to depend on a variety of causes and may have nothing to do with the condition of the stomach, but in some cases as the gastritis improves the bowels become regular.

Headache is often a symptom. It follows a variety of types, but perhaps the most common is that which comes on at intervals. In some cases it is found that whenever there is headache bile can be washed out of the stomach.

A general loss of health, of which emaciation and loss of muscular strength are the most prominent features, is found with the more severe cases of gastritis. This depends largely upon the gastritis and the interference with digestion, but in some cases it is simply due to starvation. Of their own accord, or by the advice of a physician, they give up one article of food after another until they do not get enough to eat. The exclusive diet of meat and hot water, if kept up for too long a time, has a particularly bad effect.

A considerable number of nervous and hysterical symptoms, and disturbances of sensation in the mouth, throat, and other parts of the body are often present. It is difficult to tell how far these symptoms are to be attributed to the gastritis.

*Course of the Disease.*—Chronic gastritis seems to have a natural disposition to continue and to grow worse from year to year, but with periods of remission and exacerbation. The histories, therefore, may extend over many years, and the symptoms come on in attacks. Between the attacks the patients seem to be well, but the attacks become more severe, of longer duration, and occur at shorter intervals, as the disease goes on.

I do not know of any satisfactory way of classifying the varieties of chronic gastritis. We can, however, for practical purposes, put the patients together in rather rough groups.

1. The cases in which pain referred to the region of the stomach is the principal symptom.

(a.) Pain without vomiting occurs with chronic gastritis by itself, or with retention of food, or with dilatation of the stomach, or with failure of gastric digestion. The patients do not distinguish between these conditions, they only complain of the pain, they often do not think that they have any stomach disease. The diagnosis has to be made with the stomach-tube.

(b.) Pain with vomiting occurs with hyperacidity, with dilatation, and with abnormal sensitiveness of the gastric mucous membrane to the contact of food. The last condition seems to give the most intense gastric pain there is, a pain that can only be relieved by the removal of the food from the stomach.

2. Nausea and vomiting are ordinary symptoms in all the forms of chronic gastritis, but there are some patients in whom they are particularly prominent. There are persons in whom for many years nausea, with the accompanying depression of spirits, in the early morning, is the only symptom for many years. There are persons in whom vomiting, with or without nausea, continues for years, and yet there are no other gastric symptoms.

3. There are patients who for years suffer from attacks of sick-headache, with or without vomiting. Later, instead of the sick-headaches, there are attacks of gastric pain and vomiting.

In some of the cases bile can be washed out of the stomach during every attack, in others no bile is present.

4. There are patients who suffer from retention of food in the stomach, or from dilatation without stenosis in addition to the chronic gastritis. Pain and loss of nutrition are the prominent symptoms. Treatment is often very satisfactory.

5. In the patients with dilatation and stenosis of the pylorus the pain, vomiting, and loss of nutrition get steadily worse. Treatment only relieves the pain and vomiting, the loss of nutrition continues. The question of a surgical operation regularly comes up.

6. There are patients in whom depression of spirits going on to actual melancholia is the most marked symptom. In these cases as the gastritis improves the melancholia disappears. Of course a great many of the cases of melancholia do not have gastritis at all.

7. There are patients in whom the loss of nutrition is out of proportion to the gastric symptoms. They really have a chronic gastritis, but it may be difficult to tell how far this is the cause of the loss of nutrition, and how far it is the thing to be treated. Some of these cases are very difficult to manage.

*Treatment.*—In attempting to establish a satisfactory treatment for chronic gastritis, it is important to state as clearly as possible the problem which is to be solved.

First, then, we must remember that all the patients who suf-

fer from gastric symptoms do not necessarily have chronic gastritis.

Besides those who have functional disturbance of the stomach, or cancer or ulcer of the stomach, we find many others in whom gastric symptoms are due to diseases of other parts of the body. Anæmia, uterine disease, the neurotic and hysterical condition, and constipation, often behave in this way. In old people the function of gastric digestion is often impaired simply as the result of old age. To each one of these conditions belongs its appropriate treatment, but it is not the treatment of chronic gastritis.

Still further, we must remember that in many cases of gastritis palliation of the symptoms is all that we can hope for. This is true with the gastritis associated with heart disease, emphysema, phthisis, cirrhosis, Bright's disease, gout, rheumatism, and alcoholism. It is also true of the cases in which the inflammation has gone on to the destruction of the peptic and mucous glands. After excluding all these, there remains a large and important group of cases of chronic catarrhal gastritis, in which we may hope not only to alleviate the symptoms, but to cure the disease.

It is evident, from the nature of the disease, that any treatment intended not merely to palliate, but to cure, must be of long duration, and that it must be repeated from time to time, when the inevitable relapses occur.

The different plans of treatment, then, which may be adopted are:

The curative effects of climate and mode of life.

The regulation of the diet.

The administration of drugs.

The use of local applications directly to the inflamed membrane.

It is unnecessary to lay down rules as to the sort of climate, that can be regulated by the tastes of the patient. The two points of importance are: First, the locality selected must be one where the patient can lead an out-door life. Second, the patient must live in this climate either for several years, or for a considerable part of each year.

Excellent as this method of treatment is, it is evident that it can be carried out only by a limited number of persons.

The regulation of the diet is a matter which demands consid-

eration in every case of chronic gastritis. In trying to ascertain the best way of feeding these patients, I have found only one satisfactory method, and that is to feed them experimentally with different articles of food, and then after an interval of several hours wash out the stomach and see how thoroughly these articles of food have been digested and removed from the stomach. After pursuing this course for a number of years I have arrived at the following conclusions :

It is necessary that the patient should be well fed, a starvation diet never answers.

The stomach does not require any rest from the performance of stomach digestion ; on the contrary, it is all the better for being called on to perform its natural functions.

The patients' own ideas as to what food agrees with them are usually erroneous. They are apt either to starve themselves or to select the least nutritious articles of food.

The use of artificially digested foods, or of substances, such as pepsine, to assist stomach digestion is unnecessary.

The starches, oatmeal, corn-meal, bread, the cereals, the health foods are, as a rule, bad. Portions of them remain undigested in the stomach for many hours.

Milk in adults is an uncertain article. It answers very well for some persons, not at all for others.

Meat is usually readily and well digested, but there are occasional exceptions to this rule.

Vegetables and fruits can be eaten, but the particular varieties must be selected experimentally for each patient.

I do not believe that any case of chronic gastritis is to be cured by diet alone. Even the exclusive milk diet, while it often relieves symptoms, is, as a rule, only temporary in its effects, so that the patient simply loses a certain amount of time by employing this instead of more efficacious plans of treatment.

The advantageous use of drugs belongs to the earlier stages of chronic gastritis. At that time they often palliate symptoms and sometimes even seem to cure the inflammation. In the later stages of the disease their use becomes more and more unavailing. The reliable drugs for this purpose are not numerous : the preparations of soda, potash, and bismuth, the mineral acids, glycerine, sometimes carbolic acid, sometimes iodoform, sometimes the bitter infusions. If none of these answer, it is hardly worth while to look any further. If we can combine, with the

administration of drugs, the regulation of the diet, and of the mode of life of the patient, then of course, our chances of success are much greater.

THE USE OF LOCAL APPLICATIONS DIRECTLY TO THE GLANDULAR  
COAT OF THE STOMACH.

This I regard as the most efficacious plan of treatment for those patients who are not able to leave home and seek a proper climate, but ask to be relieved without interruption to their ordinary pursuits. The local applications are readily made by the introduction of a soft rubber-tube through the œsophagus into the stomach.

Liquid applications are the best. They should be made in such quantities as to come thoroughly into contact with the entire surface of the mucous membrane, although the pyloric end of the stomach is the region where the inflammation is principally situated. They should be made at a time long enough after eating for the stomach to be as nearly empty as possible.

For many cases warm water alone in considerable quantities is the only local application needed. In some, however, there is an advantage in medicating the water, and for this purpose I employ a variety of substances.

The alkalies, the mineral acids, bismuth, carbolic acid, the salicylates, iodoform, belladonna, ipecac, gelsemium, may each one be employed according to the particular case.

For the first week it is often necessary to put the patient on a milk diet, and this can be done even with those patients who under ordinary circumstances cannot take milk at all.

Then, after a time, to the milk we add one solid meal composed of meat alone. Next, this single meal is increased by the gradual addition of fruits, vegetables, and bread. Then comes the giving of two solid meals a day, instead of one, then three solid meals, and now we get rid of the milk in part or altogether.

For the first week of this treatment it is wise not to expect any special improvement. Indeed, even a longer time than this may try the perseverance of the physician and the confidence of the patient.

Sooner or later, however, the expected improvement begins: the nausea and vomiting cease, the constipation or diarrhœa is improved; the flatulence is no longer troublesome; the head-

ache becomes less frequent ; and, of more real value than these, the improvement in the general condition of the patient becomes evident. The color, the weight, the appetite, the sleep, the spirits of the patient, all show a change for the better. Of all the symptoms, the pain is the one which is apt to persist the longest.

For two or three months the patient has to be kept under observation, and the applications to the stomach made by the physician. After this the patient is dismissed, but continues the treatment himself, first every other day, then twice a week, then once a week for several months. The regular relapses of the disease are managed in the same way, but are much more quickly relieved.

#### SUPPURATIVE GASTRITIS.

*Lesions.*—There is a suppurative inflammation beginning in the connective-tissue coat of the stomach, and extending to the other coats. This inflammation may be circumscribed, with the formation of an abscess ; or diffuse, with a purulent infiltration of the entire connective-tissue coat. The abscesses may rupture into the cavity of the stomach. The inflammation of the peritoneal coat may give rise to a general peritonitis.

*Causes.*—The disease belongs to adult life. It is more common in males than in females. There is, in some cases, a history of the over-eating of indigestible food. It evidently belongs to the class of infectious inflammations.

*Symptoms.*—The formation of the abscesses in the wall of the stomach is attended with pain in the epigastric region, vomiting, a febrile movement, and the formation of a tumor. If the abscess ruptures into the stomach, the pus may be vomited. The peritonitis often remains localized. The disease may run an acute or a chronic course.

The diffuse suppurative inflammation of the wall of the stomach runs an acute course, terminating fatally in from three to eighteen days. The patient is suddenly attacked with vomiting, pain, and tenderness over the stomach, fever, and great prostration. The symptoms of general peritonitis are soon added, and the patients rapidly get worse.

*Treatment.*—The best that we can do seems to be to alleviate the patient's symptoms by the liberal use of opium.

## ULCER OF THE STOMACH.

*Lesions.*—Ulcers of the stomach are usually single, but two or more ulcers may be formed at the same time, or successively.

Welch, from a collection of 793 cases, gives the position of such ulcers as follows :

On the lesser curvature, 288.

On the posterior wall, 235.

At the pylorus, 95.

On the anterior wall, 69.

At the œsophageal end, 50.

At the fundus, 29.

On the greater curvature, 27.

The ordinary diameter of these ulcers is from half an inch to two inches, but some are very small, and some are much larger. Two or more ulcers may become joined, and so form a large ulcerated area.

The ulcers are round or oval, largest in the glandular coat. They may destroy only the glandular coat, or the entire thickness of the wall of the stomach. The edges of the ulcers are clean cut, and their floors smooth ; but sometimes the edges are much thickened, and the floor may be formed by tissues which have become adherent. The edges and the floor are formed of amorphous granular matter, or of connective tissue.

If the patients recover, the ulcers cicatrize either with or without deformity of the stomach.

If the ulcers perforate the wall of the stomach, this perforation may be large and sudden, with the escape of the contents of the stomach into the abdominal cavity ; or the perforation may be small, and set up a localized peritonitis ; or the opening may be entirely closed by adhesions.

The ulcer may erode either the arteries or the veins of the stomach. With an ulcer there is more or less chronic gastritis.

*Causes.*—These ulcers are said to be twice as common in women as in men. They have been observed at nearly every age, but seven tenths of them are in people between twenty and forty years of age.

The ordinary explanation of the way in which these ulcers are formed is, that by embolism, thrombosis, or chronic endarteritis one of the branches of the gastric artery is occluded. The



corresponding portion of the wall of the stomach dies and is destroyed by the action of the gastric juice.

*Symptoms.*—Of all the symptoms pain is the most constant, it is absent only in exceptional cases, it varies in its quality, its intensity, its situation, and its duration.

The most characteristic pain is severe paroxysmal pain localized in a circumscribed spot in the epigastrium, coming on soon after eating and disappearing when the stomach is emptied. Of more common occurrence are paroxysms of severe pain diffused over the epigastrium and radiating in different directions. In many cases both these forms of pain exist. In the intervals between the paroxysms of pain there is often a more or less constant dull pain or feeling of discomfort at the epigastrium. The position of the pain is usually at or a little below the ensiform cartilage. It may, however, be felt as low as the umbilicus, or on either side in the hypochondriac regions.

The pain usually comes on within half an hour of taking food and continues until the stomach is emptied by vomiting or by the passage of food into the duodenum.

The pain is regularly increased by pressure over the stomach, by fatigue, and by exposure. It is diminished by rest and the recumbent position. The pain may recur at regular intervals, it may stop altogether for days or weeks.

Besides the pain belonging to the gastric ulcer the patients may also have pain due to chronic gastritis, to hyperacidity, to retention of food, to dilatation of the stomach, or to localized peritonitis.

Next to pain, vomiting is the most frequent symptom of gastric ulcer. It is apt to occur soon after taking food, or after an attack of gastric pain. The patients may only vomit occasionally, or every day, or very frequently. In some cases the irritability of the stomach is so great that no food can be retained, and the effort at vomiting will be made even when the stomach is entirely empty. The vomiting seems to be due partly to the ulcer, partly to the accompanying chronic gastritis.

It is estimated that vomiting of blood occurs in about one-third of the cases of gastric ulcer. This estimation is not of much value, on account of the occurrence of vomiting of blood with chronic gastritis and simple anæmia.

The bleeding may be preceded by pain, vomiting, and disturbances of digestion, or it may come on suddenly without any

other gastric symptoms. The quantity of blood lost may be small, or large, the larger hemorrhages are those which are most characteristic of gastric ulcer. The most important bleedings are those which are due to the erosion of a blood-vessel in the floor of the ulcer. The blood may be entirely vomited, or some of it may pass into the intestine and be discharged with the stools. With small bleedings all the blood may pass into the intestine, none of it being vomited. Rarely the patients die suddenly without any vomiting, and at the autopsy the stomach is found to be full of blood.

It is not generally understood that patients with either pernicious or simple anæmia may vomit large quantities of blood, and even bleed to death without any ulcer or erosion of the stomach. Such large bleedings, due to anæmia, and not to ulcer of the stomach, are of frequent occurrence and yet are recognized by few physicians.

The importance of a correct diagnosis is very great. If the bleeding is due to anæmia and the patients are treated for ulcer of the stomach they are very apt to die. If, on the other hand, it is appreciated that they have no stomach lesion, and they are treated for anæmia they usually recover.

It is to be remembered in this connection that many anæmic patients have pain after eating, nausea, and vomiting without any disease of the stomach.

Any of the symptoms which have been described under the heads of functional disorders of the stomach and chronic gastritis may occur in cases of gastric ulcer.

The frequency of perforation of the ulcers is variously stated by different authors. Welch gives it as six and one-half per cent. of all cases of gastric ulcer.

It is said that perforation occurs two or three times oftener in the female than in the male, and that in the female the liability is greatest between fourteen and thirty years of age. In the male there seems to be no greater liability to perforation at one age than at another. Ulcers of the anterior wall of the stomach perforate more frequently than those in other situations. The perforation may be preceded by the characteristic pain and vomiting of gastric ulcer, or only by indefinite gastric symptoms, or it may occur suddenly in a person who is apparently perfectly well.

If the perforation is such that the contents of the stomach

escape into the peritoneal cavity there is at once a severe pain and the patients pass into the condition of collapse. If they survive the shock of the perforation an acute general peritonitis is developed which is regularly fatal.

The perforation may be small and limited by adhesions. Then a localized peritonitis with collections of pus is set up around the perforation. In this way are formed the subphrenic abscesses which perforate the diaphragm and simulate empyema or pyopneumothorax.

*The Course of the Disease.*—There may be no symptoms during life, and the ulcer, or its cicatrix, is found after death from some other disease.

There are cases which last for weeks, months, or years. The symptoms are marked, but more or less severe. Some of the patients recover, others die of starvation.

There are cases in which one or more large hemorrhages form the prominent, and sometimes the only, symptom.

There are cases in which the large or small perforation of the wall of the stomach forms the prominent feature.

There are cases in which the deformity of the stomach, produced by the cicatrization of the ulcer, gives symptoms for the rest of the patient's life.

*Terminations.*—In the majority of cases gastric ulcer terminates in recovery, and such a recovery may be complete. But it may happen that chronic gastritis or deformities will be left behind which give troublesome symptoms for many years. In the fatal cases death is due to perforation, to hemorrhage, or to starvation from the inability of the stomach to retain any food.

*Treatment.*—The first point is to determine whether the patients can be fed by the stomach or by the rectum.

If they can be fed by the mouth we give, in moderate quantities and at regular intervals, milk, peptonized milk, a mixture of equal parts of milk, cream, and water, beef-juice, or Leube's or Rüdich's prepared beef.

If they have to be fed by the rectum, this is washed out once a day with warm water, and the nutrient enemata are given once in four hours. The quantity of each enema should be four ounces. They may be composed of peptonized milk, defibrinated blood, Leube's beef solution, the yolk of eggs, cream, cod-liver oil, or beef-juice. There may be an advantage in adding brandy or opium to the enema.

The drugs ordinarily employed are: The alkalies, the artificial Carlsbad salt (sodium sulphate, 5 parts; sodium bicarbonate, 2 parts; sodium chloride, 1 part; a teaspoonful in half a pint of hot water every morning), bismuth, cocaine, oxalate of cerium, nitrate of silver, iodoform, hydrocyanic acid, and opium.

In any case of suspected ulcer of the stomach with vomiting of blood, if the hæmoglobin is less than fifty per cent., and the red blood-cells less than 2,000,000 to the cubic millimetre, it is better to treat the anæmia than the ulcer of the stomach.

### CANCER OF THE STOMACH.

*Lesions.*—The new growth follows the anatomical types of colloid cancer, of cancer with cylindrical epithelial cells, and of cancer with small polygonal cells. The growth seems to originate in the glandular coat.

The most common shape for the new growth to take is that of a flattened tumor with necrotic and ulcerating centre which projects inward into the cavity of the stomach. Instead of this, however, the tumor may be quite large and of polypoid shape. In still other cases there is no tumor, but a flat infiltration which occupies more or less of the wall of the stomach. There is usually more or less chronic catarrhal inflammation of the glandular coat. The peritoneal coat is often thickened and adherent to the surrounding viscera. The process of ulceration may involve not only the new growth, but also the wall of the stomach and even extend into the adherent viscera.

While the great majority of cancers of the stomach are primary, yet secondary tumors have also been observed. Welch has collected 37 such cases, 17 secondary to cancer of the breast, 8 to cancer of the œsophagus, 3 to cancer of the mouth or nose, and the remainder to cancer of other parts of the body.

Cancer of the stomach is very often attended with the growth of metastatic tumors in other parts of the body. Welch gives the following table, based on 1,574 cases:

Lymphatic Glands.	Liver.	Peritoneum, Omentum, Intestine.	Pancreas.	Pleura and Lung.	Spleen.	Brain.	Other parts of the body.
551 35 p.c.	475 30.2 p.c.	357 22.7 p.c.	122 7.8 p.c.	98 6.2 p.c.	26 1.7 p.c.	9 0.6 p.c.	92 5.8 p.c.

*Causes.*—The disease is equally common in males and females. The maximum liability to the disease is between the ages of 60 and 70 years, but gastric cancer is common enough after the age of forty, and is even occasionally seen in persons not over 20 years old.

*Symptoms.*—The appetite as a rule is poor, either because the ingestion of food causes pain or nausea, or because there is actual distaste for food. But there are exceptional cases in which the appetite continues to be very good.

Nausea and vomiting are often present. The vomiting is of food, of brownish or yellow fluid, of coffee-ground matter, or of blood. The vomited matters may have an offensive odor. This foul smell is more constant in the contents of the stomach when they are washed out than when they are vomited. When the stomach is dilated from stenosis of the pylorus the quantity of vomit at one time is often very large. The vomiting may be an early symptom and continue throughout the disease. Or it may not come on until the disease is far advanced. Or the vomiting may accompany the early stages of the growth and then stop, not to begin again. In a moderate number of cases there is no vomiting at any time, this may be the case even when there is stenosis of the pylorus.

It is said that the situation of the cancer exerts great influence upon the frequency of vomiting, and the time of its occurrence after meals; that when the cancer involves the pyloric orifice, vomiting is rarely absent, and generally occurs an hour or more after a meal. As the stomach becomes dilated the vomiting comes on longer after a meal, sometimes not until after one or more days. Next to pyloric cancer it is cancer involving the cardiac orifice which is most frequently accompanied by vomiting. Here the vomiting occurs immediately after taking food. If there is stenosis of the cardiac orifice the food is regurgitated. When the cancer is situated in other parts of the stomach and does not obstruct the orifices, vomiting is more frequently absent. There are a great many exceptions to these rules, so many that the rules themselves are not of much practical importance.

Bleeding from cancers of the stomach is usually in small quantities, and the blood remains in the stomach long enough to be changed into coffee-ground matter, but sometimes there are very large hemorrhages.

The absence of free hydrochloric acid in the contents of the stomach in cancer of that organ is the rule. It was at one time thought that this might help in the diagnosis, but it has been found that the free acid is absent with a variety of other diseases of the stomach. Pain referred to the region of the stomach is a frequent symptom, and the pain may be very severe, but the number of patients who do not at any time have any pain is very considerable.

The presence of a tumor is the most certain evidence of gastric cancer. We look for tumors of the pylorus in the epigastric region, unless the stomach is dilated, then the tumors are found lower down in the abdomen. The tumors of the greater curvature and of the anterior wall correspond in their situation to these parts of the stomach. All these tumors are most easily felt if the patient takes a deep breath so as to depress the diaphragm. The tumors usually move pretty freely up and down with the movements of the diaphragm, but sometimes they are fastened down by adhesions. If they are close to the abdominal aorta the tumors seem to pulsate. As a rule there is no great difficulty in making out that the tumor is separate from the aorta, but sometimes it is exceedingly difficult to distinguish between cancer of the stomach and aneurism of the aorta.

Cancers of the cardiac end of the stomach cannot be felt.

The ease with which the tumor can be felt depends not only on its position, but also on its size and shape. Some of the flat infiltrations of the wall of the stomach never make an appreciable tumor. The flattened and polypoid tumors cannot be felt until they have reached some size, so that often enough we do not make out the tumor until a few months before death. If the lymphatic glands are adherent and infiltrated they increase the size of the tumor very considerably.

It is not uncommon for persons to have a chronic gastritis with its attendant symptoms for a number of years before the cancer is formed. The symptoms of the gastritis and those of the cancer then seem to make one continuous history.

In the later stages of the disease there may be a moderate rise of temperature.

The bowels are usually constipated, but there may be diarrhœa. When there is bleeding from the stomach some of the blood may come away with the stools.

The loss of flesh and of strength are sometimes the first symp-

toms to attract attention, but more frequently they come after the gastric symptoms have already existed for some time. A very considerable loss of flesh accompanying gastric symptoms always makes one think of cancer of the stomach. But it must not be forgotten that there are exceptional cases in which there is no emaciation and the general health continues to be remarkably good.

There is regularly some diminution in the quantity of hæmoglobin and the number of red blood-cells, the color of the skin changing to white or yellow. In some cases, however, these changes in the blood are as marked as they are in pernicious anæmia, and when no tumor is present the diagnosis becomes very difficult.

Late in the disease there may be œdema of the legs, ascites, and thrombosis of some of the veins, especially those of the legs.

A coma with dyspnœa, like that of diabetes, has been observed in a few cases of gastric cancer.

The ordinary duration of the disease is about one year, but some cases go on much longer—for two, or even three years.

#### COURSE OF THE DISEASE.

1. A considerable number of cases give a characteristic history—first the gastric symptoms, then the loss of flesh and strength with a tumor in the upper part of the abdomen evidently connected with the stomach.

2. Not infrequently we see patients with well-marked gastric symptoms and some loss of flesh and strength, but there is no tumor, and the diagnosis may remain doubtful for some time.

3. There are patients in whom the gradual loss of nutrition and cachectic condition are such as to make one feel sure there is a cancer somewhere in the body, but there are no local symptoms and no tumor to tell where it is.

4. There are patients who up to the time of death have the symptoms and the condition of the blood which belong to pernicious anæmia, with very little to call attention to the condition of the stomach.

5. There are cases with a primary cancer of the stomach, which is of small size and gives but few symptoms, and large secondary tumors in the liver. These patients behave as if they had cancer of the liver rather than cancer of the stomach.

6. A primary cancer of the stomach may be followed by the formation of secondary tumors in the peritoneum, with fluid in the peritoneal cavity. In some of these cases the symptoms of the cancer of the peritoneum are much more marked than those of the gastric cancer.

*Treatment.*—The patients can often be made much more comfortable, and even enabled to eat ordinary meals, by the daily washing out of the stomach. Another plan is to feed them every two hours with small quantities of easily digestible food. If the pain is bad, morphine has to be used.

A number of operative procedures have been employed by which, in some cases, life has been prolonged.

#### ACUTE CATARRHAL ENTERITIS.

The small intestine is sometimes the seat of a catarrhal inflammation which may be of mild or of severe type.

Such an inflammation is especially common in young adults, some of whom seem to have a predisposition for the disease. In some cases there is a history of indigestible food, or of exposure to the weather; in other cases no exciting cause can be discovered.

*Symptoms.*—The patients have marked and constant pain and tenderness referred to the lower part of the abdomen. There is a rapid rise of temperature from  $100^{\circ}$  to  $104^{\circ}$  F. There is marked prostration from the first, the patients being at once confined to bed. There may be vomiting, the bowels are constipated. The invasion of the disease is sudden, the patients often seem seriously ill, but yet they regularly recover. The fever subsides first, then the pain, and in one or two weeks the patients are well. Sometimes, however, the pain persists for a longer time after the subsidence of the other symptoms.

*Diagnosis.*—The disease may be mistaken for inflammation of the vermiform appendix, for peritonitis, or for gastritis.

*Treatment.*—The patients are to be kept in bed; at first on a fluid diet; when the fever has subsided, on meat principally. Continuous heat or cold should be applied over the abdomen. At first, morphine and calomel together are given in small doses, later belladonna and ipecac. The bowels are to be moved every day by enemata.



## CHOLERA MORBUS.

This name is given to an acute catarrhal inflammation of the gastro-intestinal tract, probably associated with the growth of pathogenic bacteria.

*Lesions.*—After death the mucous membrane of the stomach and intestines is found coated with mucus, congested, sometimes with little abscesses in the glandular coat.

*Causes.*—The disease is especially common in the hot weather of August and September. As exciting causes improper food and contaminated water are probable.

*Symptoms.*—The attacks are apt to come on in the night. There is first a feeling of abdominal oppression and of prostration. Then the patients empty the stomach of food and the intestines of fæces. After this there is frequent vomiting and purging of white or brownish fluid. There may be colicky abdominal pains, and painful contractions of the muscles of the abdomen and of the legs. The patients are anxious, restless, much prostrated, tormented by thirst, the skin cold, the body soon emaciated, the heart's action rapid and feeble. The patients often seem seriously ill, but yet as a rule recover. Occasionally, however, the disease proves fatal, especially in old or feeble persons.

In some cases cholera morbus cannot be distinguished from true cholera except by the absence of the cholera bacillus. The vomiting, purging, rapid emaciation, collapse, and death give the same clinical picture in both diseases.

*Treatment.*—The patients are to be kept in bed, hot fomentations are to be applied over the abdomen, and opium and stimulants are to be given according to the indications of each case. The food should be in small quantities—koumyss, beef-juice, milk, or cream and water.

## CHOLERA INFANTUM.

This name is given to a disease of young children which resembles cholera morbus, and is probably due to the growth in the intestines of pathogenic bacteria.

*Lesions.*—After death the intestines contain white or brown-

ish fluid. The mucous membrane is pale, the solitary and agminated glands are sometimes swollen, sometimes ulcerated.

*Causes.*—The disease belongs to children under two years of age. It is most apt to attack those who, from bad food, bad air, or heat, have their intestinal tract already in an unhealthy condition.

*Symptoms.*—The disease may follow an ordinary diarrhœa, or begin suddenly with purging, vomiting, and prostration. Vomiting is usually, but not always, present, and varies as to its frequency and persistency. Purging is constant and frequent. The movements are at first fecal, afterward of white or brownish fluid. The patients rapidly lose flesh and strength, the heart's action is rapid and feeble, there is constant thirst, the skin is cold, the urine is diminished. The patients are always restless and miserable, some of them have muscular twitchings, general convulsions, alternating delirium and stupor.

The disease may not last more than twenty-four hours, or it may continue for several weeks. It is always serious, and often fatal.

*Treatment.*—The children are to be fed with small quantities of koumyss, cream and water, wine-whey, beef-juice, or milk and barley-water. In the prolonged cases oil may be rubbed into the skin. The children should be kept as cool as possible, and sent to a different climate from that in which they have been taken sick. Of drugs the most reliable seem to be combinations of mercury, alkalies, and opium in small doses. Stimulants may be necessary.

#### CONSTIPATION.

In the healthy adult there should be a movement from the bowels once in each twenty-four hours. The fæces should be formed, of natural consistence and color. In some individuals the regular interval is shorter, in some it is rather longer, the character of the fæces remaining normal. If the bowels cease to move regularly, and the fecal matters accumulate in the colon, the patient becomes liable to a variety of disorders.

In the treatment of constipation it is important to determine its cause and then to manage it, principally by attention to the diet and mode of life.

In some persons constipation is merely due to the habit of only going to the water-closet when they feel the impulse to def-

ecate, whether it is once a day or once a week. In many of these patients the temporary use of a simple laxative or of a glycerine enema, with the enforcement of a regular daily hour for the defecation, is all that is necessary.

In some patients the constipation is due to chronic gastritis. If the gastritis is improved by treatment, the constipation will disappear.

The constipation may be due to an insufficient production of bile. Then we must at first use the drugs which increase the formation of bile—*ipecac*, *podophyllin*, *bichloride of mercury*, *sulphate of magnesia*, the *alkalies*, or the *mineral acids*. Later the patient must take sufficient exercise and gradually give up the use of the drugs.

The constipation may be due to general bad health or *anæmia*, and the treatment has to be directed principally to the relief of these conditions.

Improper food and drink may be the cause of the constipation. The patients require fruits, vegetables, and starches in considerable quantities. They must take sufficient water, or one of the alkaline waters, coffee, or beer.

The most difficult cases to manage are those in which the sensibility of the mucous coat of the colon is diminished and its muscular coat relaxed. In these patients we apply massage and electricity to the abdomen, enforce proper exercise and diet, and give *strychnia*, *aloes*, and *belladonna*.

In elderly persons the rectum may become filled with retained *fæces* to such an extent as to give a good deal of trouble.

In the milder cases the condition is not constant, but occurs from time to time. The patient fails to have an operation of the bowels for several days. He feels dull, languid, loses his appetite, has headache, is troubled with flatulence and uncomfortable feelings in the abdomen, which may even amount to colic. After a few days there is a slight *diarrhœa*. The passages are small, painful, do not give a feeling of relief. The patient is, at the same time, very much prostrated, vomits his food, and may even take to his bed. If you are called to attend these patients after the *diarrhœa* has begun, it is very important that you should recognize the true nature of the case. The administration of any preparation of opium, or of any drug which merely checks the *diarrhœa*, only does harm and prolongs the sufferings of the patient. A mild laxative, on the other hand, will very promptly

relieve all the symptoms. The ordinary dinner-pill is one of the best preparations for this purpose. Enemata of castor-oil and olive-oil mixed together are serviceable.

In the more severe cases the symptoms come on gradually. The patient is at first only a little constipated ; the bowels move every few days, either of themselves or with an enema, or with some laxative. And yet during this time the large intestine is not really emptied, but there is a constant accumulation of fæces in the rectum. The constipation becomes gradually more pronounced, and the patient finds that enemata and mild laxatives no longer give him a movement. Then he may use more active purgatives, which produce a number of fluid stools and yet do not empty the large intestine of the hardened fæces, which are still accumulating. So the patient goes on from bad to worse, alternating between constipation and diarrhœa, always uncomfortable, often with very severe pain in the abdomen, losing strength rapidly. If the condition is not relieved, an old person may be so reduced in this way as to die without any other disease than constipation.

The first point in the treatment is to introduce your finger into the rectum and ascertain whether or not it is filled with hardened fæces. If it is, the fæces must be scooped out with the finger or some convenient instrument, and then the rectum should be washed out repeatedly until it is entirely emptied.

After this the patient must be constantly watched and examined from time to time, to ascertain that the fæces are not accumulating again. The diet must be regulated, and aloes and strychnine may be employed to assist the action of the large intestine.

In young and middle-aged adults constipation may be prolonged for many days, and the fecal matters accumulate in the ascending and transverse portions of the colon. When this is the case the patients often become quite seriously ill. They are in bed with severe abdominal pain, an anxious face, some distention of the abdomen, and a considerable rise of temperature. Many of them look as if they had general peritonitis. In some of them the fecal tumor can be felt. The cases vary as to the relative prominence of the fever, or the fecal tumor, as symptoms.

As a rule, by the use of calomel, castor-oil, and enemata, the colon can be emptied and the patient recover.

## DIARRHŒA.

A person is said to have diarrhœa if he has every day several loose fecal or watery evacuations from the bowels.

*Causes.*—Apart from the diarrhœas which are due to inflammations or new-growths of the colon, we find the condition produced by a variety of causes.

Mental emotions often produce a transitory diarrhœa, which requires no treatment.

Extremes of heat and cold may cause a diarrhœa for which it is necessary to put the patient to bed, with a restricted diet and the use of a little opium.

Partly decayed fruit and vegetables, or undigested pieces of food in the intestine, often set up a diarrhœa. When this is the case it is necessary first to give a purgative to remove the irritative substances, and afterward to use opium.

Drinking-water which contains an excess of inorganic or organic substances is a frequent source of diarrhœa; some persons being especially susceptible to this source of irritation.

There is a form of diarrhœa in adults which is often very troublesome. In some of the patients the colon seems to be unnaturally irritable, so that improper food or atmospheric changes frequently bring on attacks of diarrhœa, abdominal pain, prostration, and mental depression, which last for a few days and then disappear.

In other cases the same symptoms—diarrhœa, abdominal pain, prostration, and mental depression—are present during so much of the time that it seems probable that the patients have a sub-acute catarrhal colitis. In these protracted cases the diarrhœa is usually in the morning, or immediately after each meal. These symptoms often continue for years, and come back repeatedly after ceasing for a time. In the severe cases there is a decided loss of flesh and strength.

An exclusive diet of milk, or of scraped beef; change of climate; cold-water enemata; castor-oil, arsenic, iron, quinine, salicylic acid, salol, and naphthaline are among our most efficient methods of treatment.

## INFANTILE DIARRHŒA.

Diarrhœa is a common disorder of infants and of young children. It is especially common during the period of dentition, in hot weather, and in bottle-fed children.

The diarrhœa may follow an acute gastritis, an acute colitis, or begin of itself. The children have every day a number of loose, bad-smelling, green, or light-colored fecal passages. There may be a febrile movement, especially in the afternoon. The patients gradually lose flesh and strength. Such a diarrhœa may last for days, weeks, or months.

*Treatment.*—If the children have a good nurse they continue to nurse. If not, they are fed on cream and water, milk diluted with a thin gruel, or koumyss. Older children can take beef-juice and scraped meat.

The medicines usually employed are: calomel, bichloride of mercury, sulphate of magnesia, castor-oil, podophyllin, rhubarb, ipecac, bismuth, and opium.

Occasionally we see cases in which there is first an ordinary diarrhœa. Then the passages become white, larger and larger, until the child passes three or four times a day enormous quantities of white, pasty fæces. The children are tormented by hunger and thirst, and become very much emaciated. They should be removed to as different a climate as possible from the one where they were taken sick. The most efficient medicine seems to be a combination of hydrochloric acid, arsenic, and opium.

## INTESTINAL COLIC.

This name is given to attacks of pain due to spasmodic contraction of the muscular coat of the colon.

Such attacks may be due to the presence in the intestine of irritating pieces of food, or fecal matter. In some persons the colon seems to be much more irritable than it is in others, so that attacks of colic are easily produced.

*Symptoms.*—The attacks are apt to come on suddenly with pain as the first symptom, and, in the mild cases, the only symptom. The pain is referred to some part of the colon, or to any part of the abdomen. Besides the pain, there may be retraction, tenderness on pressure, distention or hardness of the

abdominal wall, nausea and vomiting, a skin cold and bathed in perspiration ; rapid and feeble heart action, and a rise of temperature.

The attacks vary greatly as to their severity and duration. The mild attacks last half an hour, or a few hours, the pain is the only symptom, and it is evident that the patients are not seriously ill. The severe attacks last for days, with nausea and vomiting, a good deal of tenderness as well as pain over the colon, prostration, and sometimes a febrile movement, so that the patient may look very badly. The same patient may have one attack of colic, or several. It may be very difficult to distinguish intestinal colic from biliary colic without jaundice.

*Treatment.*—The object of treatment is to relax the muscular spasm and to remove irritating substances from the intestine. To relax the muscular spasm we use hypodermic injections of morphine and belladonna, hot fomentations over the abdomen, and hot-water enemata. To remove the irritating substances we give castor-oil or some other purgative.

In some persons the predisposition to attacks of intestinal colic exists in an unusual degree. These persons will go on year after year with repeated attacks, at first at intervals of months, then at shorter intervals, until finally the attacks may occur every day. These patients also usually suffer from flatulence and constipation. When it takes this protracted form intestinal colic becomes a serious matter. The patients lose flesh and strength, they are unable to work, the severe pain gets them into the habit of using opium.

Of these protracted cases some are easily relieved by treatment, others are very troublesome. It may be necessary to put them for a time on an exclusive diet of scraped beef, or it may be sufficient to exclude milk, tea, coffee, beer, soup, and most of the fruits and vegetables. The bowels must be kept open and sufficient exercise insisted on as soon as the patient can bear it. Opium must only be used at the time of an acute attack, it must not be continued for any length of time. The drugs which seem to be of the most service are : belladonna, ipecac, nux vomica, cannabis indica, and sodium sulphocarbolate.

## ACUTE COLITIS.

The glandular coat, the connective-tissue coat, the muscular coat, the peritoneal coat, and the lymphatic glands of the intestine are often the seat of acute and of chronic inflammation. It has been customary to include all these inflammations under the name of dysentery, or to group them under the names of catarrhal, croupous, and follicular colitis.

I think that our knowledge of the different forms of colitis is sufficient to warrant us in using more exact terms.

*Etiology.*—Sporadic cases of colitis occur in all climates. In New York they are especially common in August, September, and October. Contaminated water and food and atmospheric conditions seem to act as exciting causes. Whenever large numbers of people are brought together with irregular supplies of food, contaminated water, and imperfect sanitary arrangements colitis is likely to prevail. In every war there is always a large mortality from colitis.

Local epidemics of colitis are of frequent occurrence. Sometimes they can be accounted for by contaminated water, at other times it is difficult to account for their origin.

There can be no question that colitis is especially prevalent and frequent in tropical countries and in malarial districts.

The bacteriology of colitis is still incomplete. There can be little doubt that the *amœba coli* causes many of the cases of necrotic colitis. It is probable that the streptococci are responsible for some cases, farther than this we have no exact knowledge.

*Classification.*—A convenient classification of the different forms of colitis is as follows :

1. Acute exudative colitis.
2. Acute purulent colitis.
3. Acute productive colitis.
4. Necrotic colitis. *a.* Colitis with superficial necrosis (streptococci). *b.* Colitis with necrosis, croupous form. *c.* Colitis with deep necrosis (*amœbæ*). *d.* Colitis with deep necrosis (without *amœbæ*).



## I. ACUTE EXUDATIVE COLITIS.

*Synonyms.*—Acute catarrhal colitis, dysentery.

*Definition.*—An acute exudative inflammation involving the glandular and connective-tissue coats of the colon, attended with congestion, exudation, and an increased production of mucus.

*Lesions.*—As this form of colitis is rarely fatal in adults our knowledge of the lesions is derived from it as it occurs in children, as it is associated with other forms of colitis, as it is produced experimentally in animals, and from the character of the discharges from the bowels during life. In the more acute cases of this form of colitis the inflammation is usually confined to the lower end of the colon. The glandular and connective-tissue coats are swollen and congested and more or less infiltrated with serum and pus-cells. There is an increased production of mucus, which coats the inner surface of the colon and comes away with the stools. There may be bleeding from the surface of the inflamed mucous membrane.

In the more subacute cases the inflammation involves a considerable part of the length of the colon, there is less congestion, but the quantity of serum exuded may be large.

*Symptoms.*—In the more acute cases, when the lower portion of the colon alone is inflamed, the inflammation regularly runs its course within a week and the patients recover. The principal symptoms are the local ones: pain in the rectum, an irritability of its mucous membrane, which makes it try to discharge everything in contact with it, and the passage of small quantities of blood and mucus. Although the passages of blood and mucus are frequent, but little fecal matter comes away with them. Besides the local symptoms there is a moderate rise of temperature, with more or less prostration.

If, however, the inflammation not only involves the rectum but extends up into the colon the patients are more seriously ill, especially if they are young children. The quantity of mucus discharged from the bowels is considerable and no fecal matter may appear in the stools for one or two weeks. As the inflammation subsides the quantity of mucus decreases and the quantity of fecal matter increases. Minute examination of the stools (Councilman) shows red blood-cells, white blood-cells, epithelial cells, and bacteria. In these patients the temperature is

higher, the duration of the disease longer, and the prostration greater. Adults regularly recover, but children often die.

In the more subacute cases the inflammation may only last for a few days, but not infrequently it continues for several weeks. There is no pain in the rectum, no irritability of its mucous membrane, no small passages of blood and mucus. But during each twenty-four hours there are a number of large passages composed of fluid fæces, serum, and mucus. A single one of these discharges may measure more than two quarts. There may also be colicky pains. The patients, as a rule, are not confined to bed, although they feel weak and miserable. There is no febrile movement.

*Treatment.*—In the acute form of the disease the patients are to be kept in bed. The diet is restricted to milk, gruel, beef-juice, and scraped beef. The colon is to be emptied of fæces, by the use of castor-oil or the sulphate of magnesia. The pain is to be relieved by opium.

For the subacute cases, the treatment is to be varied with the individual. Most of the patients need opium at first. In addition to this we find that ipecac, naphthaline, salol, beta-naphthol, bismuth, subnitrate of bismuth, and castor-oil in small doses are of service, either given separately or in different combinations.

## 2. ACUTE PURULENT COLITIS.

*Synonyms.*—Acute catarrhal colitis, dysentery.

*Definition.*—An acute exudative inflammation of the colon with congestion, exudation, a large emigration of white blood-cells, and an increased production of mucus.

*Lesions.*—The changes are the same as in exudative colitis, with the exception of the large emigration of white blood-cells. These are found in large numbers infiltrating the connective-tissue coat, and the tissue between the tubules of the glandular coat.

*Symptoms.*—The clinical picture is that of an acute exudative colitis of severe type. In some cases the whole appearance of the patients is that of septic poisoning with diarrhœa. The inflammation is frequently fatal, but it is impossible to determine what the proportion is between deaths and recoveries.

## 3. ACUTE PRODUCTIVE COLITIS.

*Synonyms.*—Catarrhal colitis, dysentery.

*Definition.*—An acute inflammation of the colon attended with exudation from the vessels, an increased production of mucus, and a growth of new tissue between the glandular tubules and in the connective-tissue coat.

*Lesions.*—The inflammation, as a rule, involves a considerable part of the length of the colon. The gross appearance is that of congestion and moderate thickening, sometimes a number of small, superficial ulcers can be seen. The principal changes are in the glandular coat. We find there a growth of fibro-cellular tissue between the glandular tubules, with disappearance of the tubules. There is also a growth of cells in the connective-tissue coat. The little ulcers are formed by necrosis of small portions of the glandular coat.

*Symptoms.*—In children, who are only sick for a few days and then die from the colitis, the symptoms are those of acute exudative colitis. But after death we find that the growth of new tissue between the glandular tubules has already begun.

More frequently the inflammation continues for a longer time. The patients are ill for weeks or for months, or go on to have chronic colitis. In these long-continued cases we usually find small superficial ulcers of the glandular coat if the disease is fatal.

The patients begin with more or less abdominal pain, and a number of loose fecal passages. Besides the fecal matter, there may also be discharges of mucus and blood. Tenesmus may be present or absent.

The temperature varies very much. In some cases it will hardly be above the normal at any time, in other cases there will be a temperature of between 100° and 103° F. throughout the disease. The afternoon temperatures are the highest.

The patients are not at first very sick, but gradually lose flesh and strength, until they are glad to go to bed and stay there.

Vomiting is often present at the beginning of the disease, and there may be so much nausea as to make feeding very difficult.

As long as the colitis continues the patients have numerous fluid fecal passages, and from time to time they pass blood and mucus.

A considerable number of the patients recover, but a duration of three or four weeks is not uncommon even in the favorable cases.

*Treatment.*—The patients are to be put to bed and on a fluid diet, but in the protracted cases they must not be deprived of solid food for too long a time.

The drug which answers the best purpose is castor-oil in doses of from five to twenty drops, three or four times a day. At first opium has to be given with the castor-oil, later it is given by itself or with salol.

#### 4. NECROTIC COLITIS.

##### (a.) *Productive Colitis with Superficial Necrosis.*

This is the same as the form of productive colitis just described, the cases varying as to the extent of the necrosis.

##### (b.) *Croupous Colitis.*

*Lesions.*—The mucous membrane is congested, swollen, infiltrated with fibrine and pus, and a layer of fibrine and pus is formed on its free surface. There is superficial or deep necrosis of the inflamed portions of the colon. The inflammation is sometimes diffuse, more frequently confined to a number of small, circumscribed portions of the mucous membrane. The inflammation may involve the rectum alone, or the rectum with more or less of the colon. After the inflammation has subsided superficial or deep ulcers are left, according to the extent of the necrosis.

This form of colitis is always severe, and often fatal. It behaves as do the infectious inflammations.

*Symptoms.*—The patients have numerous small, painful passages of blood and mucus. If the inflammation extends above the rectum they also pass large quantities of brownish fluid. There is nearly constant pain, irritability, and a desire to go to stool. There may also be colicky pains, strangury, vomiting, and jaundice. There is regularly a febrile movement, sometimes preceded by chills, throughout the disease; but in some of the bad cases the temperature is low throughout. In the severe cases the prostration is marked, the heart's action is

rapid and feeble, delirium, stupor, or convulsions are developed. The disease lasts for from one to six weeks.

*Prognosis.*—Croupous colitis is a very serious disease. It is often fatal ; it is often followed by chronic colitis. It is especially fatal in old persons, in young children, in tropical countries, with epidemics, with inflammation of the entire length of the colon.

*Treatment.*—The patients are to be kept in bed and on a fluid diet ; they may require stimulants. Of medicines, the most efficient are castor-oil, sulphate of magnesia, ipecac, salol, and opium. The castor-oil and sulphate of magnesia are given at long intervals and in full doses, to empty the colon of fæces. They are given in small doses, at short intervals, to lessen the colitis. The ipecac is given once or twice a day, in doses of from ten to twenty grains. The salol is given in five-grain doses at intervals of from one to four hours. The opium is given to relieve pain.

#### (c.) *Amœbic Colitis.*

This is a severe form of necrotic colitis apparently due to the presence in the wall of the colon of the amœba coli. It seems to be especially common in tropical countries, but is not rare in temperate climates. The method of infection has not been determined.

*Lesions.*—A considerable part of the whole length of the colon is usually involved.

The amœba coli is a single spheroidal cell, from five to eight times the diameter of a red blood-cell, with granular protoplasm and a vesicular nucleus. It often contains vacuoles. When seen alive on the warm stage it will change its shape and position. It is found in the stools, in the connective-tissue coat of the colon, in abscesses in the liver, and in abscesses in the lungs. A great number of micrococci and of bacilli of different kinds are also found in the stools and in the intestinal ulcers.

The morbid process seems to begin in the form of circumscribed thickening, infiltration, and necrosis of portions of the connective-tissue coat of the colon. These changes are apparently due to the presence of the amœba coli. The necrosis soon extends to the glandular coat, and ulcers are formed which extend down to the muscular or peritoneal coats and have overhanging edges. In addition there are more or less infiltration of the walls and floors of the ulcers with pus-cells, cell growth

between the glandular tubules, and increased production of mucus in the tubules which are left. These additional changes seem to be due to the mixed infection by the other bacteria which are present in the intestine. The cases vary as to the size and number of the ulcers. In the bad cases nearly the whole of the glandular coat of the colon is destroyed.

In a considerable number of cases of amœbic colitis there are abscesses in the liver. There may be one or two large abscesses, or a great many small ones. There is first necrosis of portions of the liver, then softening and breaking down, and then more or less suppuration of the walls of the cavities.

Similar necrotic abscesses containing amœbi may be found in the right lung.

*Symptoms.*—The invasion of the symptoms is either acute or gradual.

In the acute cases the patient is suddenly attacked with colicky pains and diarrhœa, often with nausea and vomiting, and sometimes a rise of temperature. The stools are frequent, fluid, and sooner or later contain blood and mucus. The quantity of blood may be considerable.

In the more gradual invasion there is at first nothing but a painless diarrhœa—several loose, fecal stools in the twenty-four hours.

The patients have no appetite, they lose flesh and strength, and become anæmic. In some of them the symptoms are continuous, in others there are remissions and exacerbations. Although the temperature may reach 104° F., the febrile movement is not a feature of the disease. The most noticeable symptoms are the frequent stools of fluid fæces, bad-smelling fluid, mucus, and blood, the pains in the abdomen, and the progressive loss of flesh and strength. The disease may only continue for two weeks, or it may be protracted over several months. The same patient may have several attacks of the disease.

A serious complication is the formation of the amœbic abscesses of the liver, which may occur at any time in the course of the colitis, either in mild or severe cases.

Of less frequent occurrence are the amœbic abscesses in the lungs, with cough and bloody, foul-smelling expectoration.

I have seen one case in which the pulmonary symptoms preceded the diarrhœa by three days.

Perforation of the intestine followed by collapse or by peri-

tonitis sometimes occurs. There may also be peritonitis without perforation.

*The prognosis* is serious, not only from the extent of the destruction of the wall of the colon, but from the liability to abscess of the liver, abscess of the lung, and peritonitis.

*Treatment.*—While the disease is active the patients are kept in bed and on a fluid diet; they may require alcoholic stimulants.

For the pain and sleeplessness, small doses of some preparation of opium are necessary.

For the colitis ipecac in large doses, or castor-oil in small doses, seems to be the most useful drugs.

Large enemata of solutions of bichloride of mercury 1 to 5,000, and of quinine 1 to 5,000, have been employed.

*d. Colitis with Necrosis and Suppuration without Amœbæ.*

I have seen several fatal cases of which the symptoms were the same as those of amœbic colitis, but there were no abscesses of the liver or lung, no amœbæ in the stools or in the wall of the intestine, but very extensive necrosis and suppuration of the connective-tissue coat of the colon.

I have also seen patients who recovered from an illness lasting for several weeks and resembling amœbic colitis, but without any amœbæ in the stools.

CHRONIC CATARRHAL COLITIS.

This regularly follows an acute or subacute productive colitis.

*Lesions.*—The rectum alone is the portion of the colon which is most frequently involved in chronic inflammation, but the rest of the colon is also liable to the same changes. There is regularly an increased quantity of mucus produced by the mucous glands; thickening of the glandular coat alone, or of all the coats of the colon; sometimes ulceration of the glandular coat, sometimes little polypoid growths on the surface of the mucous membrane. It is surprising to find how little anatomical change in the mucous membrane is associated with a fatal diarrhœa.

*Symptoms.*—The cases vary as to the severity of the inflammation, the extent of colon inflamed, and as to whether the colitis is chronic from the outset, or succeeds an acute colitis.

In the mild cases the general health may remain fairly good,

although the patients pass every day more or less mucus, and occasionally a little blood. In the severe cases, besides the mucus and blood there are numerous discharges of fluid fæces, the patients lose flesh and strength, and finally may die exhausted by the disease.

*Treatment.*—The patients do best in a dry inland climate. The food must be restricted, even an exclusive milk diet may be necessary. The best medicines are castor-oil in small doses, the mineral acids, ipecac, naphthalin, and salol.

#### MEMBRANOUS ENTERITIS.

This is a chronic catarrhal colitis attended with a large production of very tenacious mucus, which comes away with the stools in the form of masses, cords, or tubes looking like membranes.

*Causes.*—The disease belongs to middle life, and is more common in women than in men.

*Symptoms.*—So far as the colon is concerned, the symptoms are: constipation, sometimes diarrhœa, flatulence with its attendant pains, and the passage at varying intervals of the so-called membranes already mentioned.

But with these symptoms are usually associated complicating conditions which are often more distressing than the intestinal symptoms. There may be gastric dyspepsia, functional disorders of the liver, diseases of the uterus and ovaries, hysteria, or hypochondriasis.

*Prognosis.*—The disease is not often a fatal one, but yet a considerable time may elapse before the patient entirely recovers.

*Treatment.*—In the more severe cases the patients must be put on milk diet, or the use of scraped beef and hot water. An out-of-door life in a dry inland climate is regularly of service.

The most useful drugs are the alkalies, the mineral acids, small doses of castor-oil, ipecac, and sometimes small doses of opium. There is also sometimes an advantage in the use of large enemata of cold water, and of massage of the abdomen.

#### CHRONIC COLITIS WITH ULCERS.

*Lesions.*—The ulcers are small or large, numerous or few, close together or scattered, confined to the rectum or extending up on the colon. The mucous membrane between the ulcers is more



or less changed by catarrhal inflammation. The walls of the floors of the ulcers are formed of granulation tissue.

*Causes.*—The ulcers are regularly formed during the course of an acute necrotic colitis.

*Symptoms.*—The patients have frequent passages of blood, of mucus, or of fecal matter. They steadily lose flesh and strength.

*Treatment.*—The patients are to be managed in the same way as those who suffer from catarrhal colitis. But if the ulcers are situated low down in the rectum, there is also an advantage in the use of local applications to the ulcers. These applications may be made by suppositories, or by enemata, or the patient is etherized and the ulcers brought into view with the speculum.

### CARCINOMA OF THE COLON.

The new-growth usually follows the type of colloid cancer, or of glandular cancer with cylindrical cells. The favorite situations are the rectum and the caput coli. The new-growth surrounds the gut and extends longitudinally for one or two inches; less frequently it involves a considerable part of the length of the colon. The growth may cause a stricture of the colon, or may project inward in soft masses, or may ulcerate. It often infiltrates the surrounding tissues, it may cause a local peritonitis, or the ulceration may go on to perforate the wall of the colon.

*Causes.*—The disease occurs regularly in persons over thirty years of age.

*Symptoms.*—If the growth is situated in the rectum the first symptoms are constipation, flatulence, pain, and constricted stools; or simply a number of small fecal passages every day. The patients complain of the annoyance of having to defecate so frequently instead of having a single large movement. In other patients there is at first a diarrhoea, the passages are fluid, and more or less blood and mucus are also passed. At first, and even for a number of months, the general health suffers very little.

As the disease continues the local symptoms become more marked, and the patients lose flesh and strength and become cachectic. The tumor can be felt in the rectum as a hard ring constricting the gut, or as a hard diffuse mass about the gut, or as a soft tumor projecting inward. As the disease goes on the

stricture becomes tighter and tighter ; or there is paralysis of the sphincter with a constant flow of brown, sanious fluid.

The patients die worn out with the disease, or with retention of fæces, or from perforation of the intestine, or from peritonitis.

If the growth is situated in the caput coli the first symptoms are gradual loss of flesh and strength, flatulence, alternating diarrhœa and constipation, nausea, and vomiting. Or the first symptom is pain coming on in attacks, first at long intervals, then more and more frequently. These symptoms continue, the patients lose flesh and strength, and become cachectic ; there may be distention of the small intestine. The tumor can be felt in the right side of the abdomen, when it has reached a sufficient size.

Occasionally we see cases of cancer of the rectum, or of the caput coli, in which there are no local symptoms. The only evidences of the disease are the loss of flesh and strength, and the tumor.

*Treatment.*—The only efficient treatment is the removal of the tumor by a surgical operation.

## THE LIVER.

THE liver occupies the right hypochondriac region, and extends toward the left into and a little beyond the epigastrium. Its upper surface is in contact with the diaphragm, its lower surface touches the stomach and the colon. The upper border of the liver is at the level of the fourth or fifth rib in the mammary line, at the level of the seventh rib in the axillary line, at the level of the eighth or tenth rib behind. The upper border is overlapped by the lungs down to the sixth rib in front, and to the tenth rib behind. The lower border comes down to the free edge of the ribs in front. The fundus of the gall-bladder touches the anterior abdominal wall immediately beneath the free border of the ribs opposite the tip of the tenth costal cartilage.

To determine the size of the liver, the patient is placed on his back. Percussion is made from above downward in the mammary line, and should give dulness at the fourth or fifth rib, flatness at the sixth rib, and from thence down to the free border of the ribs. Percussion in the axillary line should give flatness at the seventh rib. The lower border of the liver is made out by percussion and palpation, the latter is the more certain. In practising palpation the patient should breathe deeply so as to depress the diaphragm and liver. It is easy to mistake the right rectus muscle for the right lobe of the liver. Another method of palpation is to make the patient sit bending forward and resting his arms on a chair. The physician sits behind the patient and examines through the relaxed abdominal wall.

As regards the component parts of the liver: the hepatic cells are liable to a variety of degenerative changes; the connective-tissue stroma may be the seat of exudative, suppurative, productive, or necrotic inflammation; the blood-vessels may be occluded, obliterated, or inflamed; the bile-ducts may be obliterated, or inflamed, or may contain calculi.

Of the functions of the liver the two which principally concern us are: the production of bile, and the chemical changes effected in the blood coming from the portal vein.

1. *The Production of Bile.*—This fluid is produced at the rate of about forty ounces in each twenty-four hours. It passes into the duodenum, thence into the intestinal tract, where part remains, and part is reabsorbed by the lymphatics of the wall of the intestine. The portions which remain in the intestine assist in the digestion of fat and peptones, act as an antiseptic to prevent the decomposition of the contents of the intestine, assist in exciting the peristaltic action of the colon, and are finally discharged mixed with the fæces.

2. *The Chemical Changes Effected in the Blood.*—The blood from the abdominal viscera, charged with the matters absorbed from the intestine, passes through the entire venous system of the liver. While it does this the matters contained in it undergo chemical changes and are separated into excrementitious substances which are eliminated by the kidneys, and other substances which circulate in the blood and nourish the tissues.

#### FUNCTIONAL DISORDERS OF THE LIVER.

The functions of the liver may be disordered either with or without structural disease of this organ. These disorders may occur by themselves, or be associated with disturbances of the stomach and of the intestines.

The cases may be arranged in two groups:

##### I. DISTURBANCES OF THE BILE-PRODUCING FUNCTIONS OF THE LIVER.

The quantity of bile produced is too small. This is followed by imperfect digestion of fats and peptones and consequent loss of nutrition; by decomposition of the matters contained in the intestine, with flatulence and possible poisoning; and by constipation.

The patients become anæmic and emaciated, or are rather fat, but yet feeble and flabby. They are constipated, with flatulence and its associated pains. The fæces are hard and light colored. The tongue is coated, the mouth and throat are dry, with a bitter taste. The skin is pale and muddy. The appetite is poor and capricious. The urine contains an excess of oxalate

or phosphate of lime. There are headache, mental lassitude, and abnormal sensations in different parts of the body. In the bad cases hypochondriasis, neurasthenia, hysteria, or melancholia may be developed.

*Treatment.*—The object of treatment is to increase the production of bile, and this is to be effected by improving the general health, and by using remedies which will increase the production of bile.

In the milder cases the patients should eat meats, vegetables, and fruits, take exercise in the open air, and one hour before meals take a tumbler of hot water with a small quantity of sulphate of magnesia. Or, instead of this, aloes, podophyllin, rhubarb, ipecac, phosphate of soda, sulphocarbonate of soda, bichloride of mercury, or the mineral acids, may be given separately or in various combinations.

In the more severe cases the patients may be so feeble that they require rest in bed and massage for a time before they can begin to take exercise.

Some of the patients are benefited by cold-water douching and by the use of iron.

## 2. IMPERFECT CHEMICAL CHANGES IN THE BLOOD WHICH PASSES THROUGH THE LIVER.

These patients seem to be able to digest all kinds of food, for their nutrition does not suffer, and they are often large, strong, florid persons. Not infrequently they habitually eat and drink too much. They may have the gouty diathesis.

The most marked symptoms are : Feelings of fulness in the head, vertigo, loss of memory, abnormal sensations in different parts of the body, hypochondriasis, sometimes morning diarrhœa. The urine is scanty and high colored, it contains an excess of uric acid and of urates. It may contain small quantities of albumin and of sugar.

*Treatment.*—The patients should avoid the use of tobacco, wines, spirits, sugars, and starches. They should take active exercise. They often get temporary relief by the use of purgatives. They may be much benefited by the regular course of treatment at Carlsbad.

## JAUNDICE.

This name is used to designate the staining of the skin and many of the tissues and fluids of the body by the coloring matter of the bile.

The pigment is found in the urine ; it stains the conjunctiva, skin, mucous membranes, the sweat, and the discharges from wounds.

It has been customary to distinguish two forms of jaundice :

1. Obstructive, or hepatogenous, jaundice, caused by an obstruction of the hepatic or common bile-duct. The bile accumulates in the bile-ducts within the liver, is reabsorbed from the biliary passages and circulates in the blood. 2. Non-obstructive, or hæmatogenous jaundice. In this form of jaundice it was supposed that the bile is formed, passes into the intestine, is reabsorbed into the blood ; but after being reabsorbed into the blood the regular chemical changes which dispose of the coloring matter of the bile do not take place. So this coloring matter circulates unchanged in the blood throughout the body.

At the present time the disposition is to hold that all jaundice is caused by obstruction, but that the obstruction may involve the large bile-ducts without the liver, or the small ducts within the liver.

1. Obstruction of the bile-ducts outside of the liver (the hepatic and common ducts) is usually caused by :

Biliary calculi, inspissated bile, or foreign bodies from the duodenum.

Inflammation of the common bile-duct or of the duodenum.

Congenital malformations of the bile-ducts, perihepatitis, ulcers of the duodenum.

Tumors of the bile-duct, or of the duodenum.

Pressure on the bile-ducts by tumors in the abdominal cavity.

2. Obstruction of the bile-ducts within the liver is seen with : Cirrhosis of the liver, fatty liver, and tumors of the liver.

The infectious diseases, especially their more malignant forms.

The severity of the symptoms of jaundice are in proportion to the quantity of bile-pigment which accumulates in the blood and tissues, and the length of time during which the jaundice continues.

The mild and temporary forms of jaundice give no symptoms.

The well-marked forms, lasting for a number of days, are accompanied by: A deep yellow color of the skin and mucous membranes, constipation, the fæces white or clay colored, the urine of a dark yellow color, the mind rather dull and apathetic, sometimes headache, sometimes a rise of temperature, the pulse full and slow, loss of appetite, sometimes nausea and vomiting, more or less loss of strength, itching of the skin.

The severe cases, in which the jaundice increases and cannot be relieved, pass into the condition of alternating stupor and delirium, lose flesh and strength rapidly, bleed into the skin and from the mucous membranes and finally die.

#### SIMPLE, OR CATARRHAL JAUNDICE.

*Lesions.*—There is a catarrhal inflammation of the common bile-duct alone, or in addition there is a catarrhal gastritis, or a catarrhal duodenitis. The inflammation is attended with so much swelling of the mucous membrane of the common bile-duct as to obstruct the flow of bile from the liver into the intestine.

*Symptoms.*—The patients suffer from general malaise, headache, dulness, sleepiness, or vertigo. The tongue is coated, there is loss of appetite, nausea, and vomiting. There may be a febrile movement. Sometimes there is pain over the duodenum. After one or more days comes the yellow color of the conjunctiva and of the skin, the clay-colored fæces, and the dark-colored urine, with the itching of the skin. The urine, besides the bile pigment, contains albumin and casts. After the appearance of the jaundice the gastric symptoms may subside.

If the bile-duct alone is inflamed the cases run a mild course. The one symptom is the jaundice. The patients are not confined to bed, the cerebral symptoms are slight, there is no vomiting and no fever.

If the duodenum is inflamed as well as the common bile-duct the prostration is more marked. There is often a good deal of pain in the epigastrium. Nausea, vomiting, and a febrile movement are absent, or not severe.

If the stomach also is inflamed the condition of the patients is much worse. They are often confined to bed. There may be stupor and delirium. Nausea, vomiting, and a febrile movement are regularly present. The symptoms of the gastritis may precede by several days the appearance of the jaundice.

When gastro-duodenitis with jaundice occurs in a person who already has a cirrhotic or a fatty liver, it is often fatal.

*Treatment.*—While the symptoms are active the patient is kept in bed and on a fluid diet, with the addition of sodium bicarbonate to the milk. As the gastric symptoms subside they get back to solid food, even when they are jaundiced. The jaundice is treated by enemata. Either a quart of ice-water is slowly introduced into the rectum and then passed; or a quart of hot water is used in the same way. These injections are given once or twice a day.

Instead of this an enema of half a pint of olive-oil and half an ounce of castor-oil is given every day and retained for as long a time as possible.

#### CHRONIC JAUNDICE.

Although cases of simple jaundice usually run their course within a few weeks and recover, yet they sometimes continue for a longer time. In these protracted cases the disease behaves in one of three ways:

1. All the symptoms continue with a steadily increasing jaundice. The patients lose flesh and strength, there are bleedings from the mucous membranes and in the skin. The patients die after several months, emaciated and deeply jaundiced.

2. The patients suffer from repeated attacks of gastro-duodenitis and jaundice. Between the attacks they seem almost well, but yet there is always a little bile in the urine. It is difficult to distinguish these cases from the cases with a movable calculus in the common bile-duct.

3. The jaundice continues, but does not increase. The patient's general health remains comparatively good. After a number of months the jaundice finally disappears.

#### ACUTE DEGENERATION OF THE LIVER.

*Synonyms.*—Acute yellow atrophy of the liver. Malignant jaundice. As the result of some unknown poison there is a very rapid and fatal degeneration of the hepatic cells and the renal epithelium.

*Lesions.*—The liver may be at first somewhat enlarged and then become smaller, or it becomes smaller from the first. The diminution in size is very rapid and goes on until the liver only



weighs one or two pounds. These very small livers are yellow and flabby.

In other cases the liver is not much diminished in size, its consistence is firm, its color is mottled red and yellow.

In all cases there are profound changes in the hepatic cells. They become swollen, coarsely granular, and finally disintegrate and break down.

The spleen is large and soft. The stomach is the seat of acute catarrhal inflammation.

In the intestines the solitary and agminated glands are swollen.

The kidneys are the seat of very intense acute degeneration. In most of the tubes the epithelial cells are dead and detached from the walls of the tubes.

The muscles of the heart undergo fatty degeneration.

There are extravasations of blood in the skin, the mucous membranes, and the serous membranes.

*Causes.*—The disease has been observed in persons between the ages of one and sixty-nine years, but it is especially common in persons between twenty and thirty years of age. It is more common in women than in men. It has been seen a number of times in pregnant women.

*Symptoms.*—The invasion of the disease is gradual, or sudden.

If it is gradual, the patients suffer from nausea, occasional vomiting, general malaria, sometimes jaundice for from three days to three weeks.

If it is sudden, the patient at once becomes seriously ill with a rapid development of all the symptoms of the disease.

When the disease is established, there is frequent vomiting, first of food, then of brownish fluid, coffee-ground matter and blood. There is a diarrhœa with clay-colored fæces, and later bleeding from the intestines. Jaundice in two-thirds of the cases appears early in the disease, in one-fourth of the cases from the fifth to the seventh day, in a few cases not until one or two days before death. In very rapid cases there may be no jaundice. There may be pain over the region of the liver. There is a febrile movement during the active period of the disease, but the temperature runs an irregular course, sometimes very high, sometimes falling below the normal. The pulse becomes rapid and feeble.

There are cerebral symptoms—headache, alternating stupor and delirium, general convulsions, and coma.

The tongue becomes brown and dry, the prostration is very marked.

The liver may at first be enlarged and then become smaller, or it may become smaller from the outset. The diminution in size, as we mark it out by percussion, is very rapid, and finally the liver dulness may entirely disappear. In some cases, however, the liver remains throughout the disease of nearly normal size.

The urine contains bile-pigment, albumin, casts, blood, leucin, and tyrosin. The urea and uric acid are diminished in quantity.

There are often hemorrhages from the stomach, intestines, and kidneys, and in the skin.

The ordinary duration of the active symptoms is a week, but a prolonged period of invasion may lengthen the whole time of the disease to four weeks, and the bad cases may die within twelve hours.

*The prognosis* of the disease is very bad.

#### CIRRHOSIS OF THE LIVER.

*Synonym.*—Chronic interstitial hepatitis.

*Lesions.*—There is a chronic productive inflammation of the stroma of the liver with more or less degeneration of the hepatic cells. The new tissue follows the distribution of the stroma of the liver. It surrounds large or small groups of acini, or grows diffusely between the liver-cells, or is in the form of large bands and masses. According to the distribution of the new connective tissue, the liver is coarsely or finely nodular, or smooth, or lobulated. The liver-cells may become atrophied or fatty. The small veins and bile-ducts are often obliterated. The larger bile-ducts may be the seat of catarrhal inflammation. The liver remains of normal size, or becomes small or large. We find after death: small nodular livers, small smooth livers, large nodular livers, large smooth livers.

The obstruction to the passage of the blood from the portal vein through the liver causes the spleen to become large and hard, the mucous membrane of the stomach and small intestines to be congested and coated with mucus, the peritoneal cavity to contain serum.

*Causes.*—The disease belongs to adult life, and is usually caused by alcohol or syphilis. It is, however, occasionally seen in children, and may occur without discoverable cause.

*Symptoms.*—These depend on :

1. The disturbance of the bile-producing function of the liver, with the consequent digestive disturbances and loss of nutrition.
2. The accompanying chronic gastritis.
3. The interference with the passage of the blood through the liver and the consequent dropsy.
4. The development of jaundice.
5. The existence of a complicating nephritis, arteritis, or emphysema.

*Physical Signs.*—These depend on the changes in the size of the liver and the smooth or nodular character of its surface ; on the enlargement of the spleen ; and on the presence of fluid in the abdominal cavity.

*The course of the disease varies in different persons.*

1. There are persons who have cirrhosis of the liver for a number of years without any symptoms to call attention to that organ.
2. In a good many cases of cirrhosis the principal symptoms are those of the accompanying chronic gastritis. This gastritis seems to be partly due to chronic alcoholism, or some other ordinary cause, partly to the chronic congestion caused by the cirrhosis. It behaves like any chronic gastritis, but is often severe and rebellious to treatment, and the patient may vomit blood in large or small quantities.
3. The anatomical changes in the liver after a time affect the functions of the organ in such a way that the patients steadily lose flesh and strength. This loss of nutrition is a very constant symptom, and seems to have a large share in causing the death of the patients. It is most frequently associated with dropsy, but may exist and cause death without any dropsy at all.
4. Ascites is one of the ordinary features of cirrhosis ; general dropsy occurs less frequently. When the dropsy has once appeared it is apt to continue, and the patients get steadily worse. But occasionally even a large abdominal dropsy can be gotten rid of and the patient do well for a number of years.
5. Patients with cirrhosis of the liver are liable to slight attacks of catarrhal jaundice, and to severe attacks of gastro-

duodenitis with jaundice. These latter attacks are attended with fever and cerebral symptoms and are often fatal.

6. With the large, nodular, cirrhotic livers (hypertrophic cirrhosis) there often occurs a very fatal form of jaundice. It behaves like a bad obstructive jaundice, although the hepatic and common bile-ducts are pervious.

The patients have pain over the liver, vomiting, constipation, a constantly increasing jaundice, a febrile movement, gradual emaciation, and cerebral symptoms. All of them die.

*The prognosis* of cirrhosis is unfavorable, but yet in many cases the course of the disease is slow, and in some it is arrested altogether. The patients have no symptoms, although the cirrhosis still exists.

*Treatment.*—In many cases of cirrhosis we only treat the chronic gastritis. This is done in the usual way by regulation of the diet and mode of life and by lavage. But it is found that if the cirrhosis progresses it becomes more and more difficult to control the gastritis.

In the cases with marked loss of nutrition the use of alcohol and of tobacco is to be prohibited. The patients should lead, as far as possible, an out-door life. It may be necessary to relieve constipation and to give drugs which increase the production of bile.

If there is a distinct history of syphilis it may be necessary to give mercury and the iodide of potash.

Moderate quantities of fluid in the peritoneal cavity can be gotten rid of by diuresis and by purging, but when the abdomen is distended with fluid it is necessary to tap.

#### ABSCESS OF THE LIVER.

Suppurative inflammation of the liver may be produced by a similar inflammation of the walls of the bile-ducts or of the veins within the liver.

Small abscesses are formed in the liver by the lodgement of infectious emboli in the vessels.

Most of the abscesses in the liver are due to the presence and growth of the *amœba coli*. Whether abscesses are produced by traumatism, or by the ordinary bacteria of suppuration, is uncertain.

*Lesions.*—The abscesses usually follow one of four anatomical forms.

1. They resemble ordinary abscesses in any part of the body. There are one or more abscess cavities of considerable size, filled with well-formed pus, and their walls infiltrated with pus-cells. The abscesses may be situated in any part of the liver. This is the least fatal form of liver abscess.

2. There are one or more cavities in the liver with ragged, irregular, necrotic walls, and containing a thin reddish, or brownish, or yellow fluid, with fragments of dead liver-tissue, but very few pus-cells.

3. A considerable area of liver-tissue is dead, with little cavities filled with pus or puriform fluid scattered through it.

4. The entire liver is studded with small abscesses, some of them too small to be seen with the naked eye.

In all these abscesses the *amœba coli* may be found. In the smallest abscesses the first change is a necrosis of the liver-tissue. Whether the different anatomical characters of the abscesses are due to the addition of bacterial infection to the amœbic infection has not been worked out.

If the abscesses are situated near the surface of the liver, there will be exudative inflammation and the formation of adhesions.

If left to themselves the abscesses usually increase in size until they perforate through the diaphragm into the lung, the pleura, or the pericardium; or into the stomach, the intestines, the peritoneal cavity, or the pelvis of the right kidney; or through the anterior abdominal wall.

Even without perforation there may be inflammation of the right pleura, or abscesses in the right lung.

*Symptoms.*—1. As it is observed in tropical countries. The invasion is, or is not, preceded by the symptoms of colitis. It is acute, ushered in by chills and a febrile movement and marked prostration.

The chills may be repeated throughout the disease. The fever is of the continuous, remittent, or intermittent type, and is accompanied by sweating. There are pains and tenderness over the liver.

Jaundice is not a constant or marked symptom.

Nausea and vomiting are often present.

There may be localized, or general, peritonitis. There may be ascites without peritonitis.

Perforation of the abscess is followed by symptoms according to its position.

2. As it is observed in New York. The invasion is gradual. There is sometimes a history of colitis occurring just before the symptoms of the abscess, or preceding these symptoms by several weeks or months.

The patients feel chilly and feverish, lose their appetites, feel sick, may have nausea and vomiting, but often do not go to bed for several days. After they get to bed the fever continues, with chills and sweating. The nausea and vomiting continue, they lose flesh and strength, there are pain and tenderness over the liver. If perforation, or death, or recovery do not take place these symptoms may continue for weeks, months, or years.

The physical signs vary with the position and size of the abscess. Large abscesses in the lower part of the right lobe of the liver, or in the left lobe, cause a considerable enlargement of the liver downward into the peritoneal cavity.

Abscesses in the upper part of the right lobe of the liver projecting up into the right pleura may give no downward enlargement of the liver at all. There is only dulness over the lower part of the right lung behind, and this dulness is more marked if there is fluid in the pleural cavity.

Some abscesses in the right lobe only give a fulness of the right hypochondrium without enlargement of the liver either upward or downward. With small abscesses there may be no enlargement of the liver at all.

*Treatment.*—The success of treatment depends on the position and character of the abscess. If of moderate size and a single cavity containing pus it can be cured by aspiration. Larger abscesses need to be opened and, if they project into the pleura, this may be difficult. Multiple abscesses and diffuse necrosis are not amenable to treatment.

#### THE FATTY LIVER.

*Lesions.*—There is a deposit of fat in the liver-cells, with an increase in the size of the liver. There are no changes in the stroma or in the blood-vessels. The larger bile-ducts may be the seat of acute, or of chronic, catarrhal inflammation.

*Causes.*—The disease occurs at all ages. It is more common in women than in men. It seems to be caused by alcoholism, over-eating, want of exercise, and by phthisis and other wasting diseases.

*Symptoms.*—1. Many fatty livers give no symptoms at all.

2. With some fatty livers there are marked symptoms of disturbance of the functions of the liver, with the physical signs of a large liver. The patients are fat, but anæmic and feeble. Mental lassitude and depression, and abnormal sensations in different parts of the body are prominent symptoms. The bowels are constipated and the functions of the stomach are deranged. There is often added a chronic gastritis, sometimes with vomiting of blood.

*The treatment* of these cases is by regulation of the diet and mode of life, and the use of drugs which increase the production of bile.

3. The infiltration of the liver-cells with fat may be unusually great, and the increase in the size of the liver enormous. In these patients the first symptoms are those of disturbance of the bile-producing function of the liver, and of chronic gastritis. These symptoms do not improve, but get worse. The patients become so feeble and anæmic that they are confined to bed. They become a little jaundiced. They finally die in a condition of extreme exhaustion.

4. Patients with fatty livers and chronic gastritis are particularly liable to attacks of acute gastro-duodenitis with jaundice. These attacks are severe, with a good deal of fever and delirium, and often prove fatal.

5. Patients with large fatty livers may develop an increasing and fatal jaundice without obstruction of the hepatic or common bile-duct. The jaundice behaves like that which accompanies hypertrophic cirrhosis.

#### THROMBOSIS OF THE PORTAL VEIN.

*Lesions.*—The main trunk of the portal vein, or one of its branches, is partly or completely filled by a thrombus. This thrombus does not soften, is not infectious, but is replaced by new connective tissue so that the vein is permanently obstructed.

*Causes.*—Cirrhosis or cancer of the liver, chronic peritonitis, tumors of the abdominal cavity, and injuries inflicted over the

upper part of the abdomen act as causes. There are also cases which occur without discoverable cause, especially in old persons.

*Symptoms.*—First there may be the symptoms of the cirrhosis, the cancer, the chronic peritonitis, the tumors, or the injuries.

The symptoms of the thrombosis are developed rapidly. There is rapid and persistent dropsy of the peritoneal cavity. The spleen is enlarged. There are vomiting of food and of blood, diarrhœa, and bleeding from the intestines. The urine is scanty.

In some of the patients the symptoms continue without interruption. They become very feeble, delirious, and unconscious. Death takes place within a few days. In these patients there may be no dropsy. In other cases, where the dropsy is the principal feature without much bleeding or prostration, the patients may live for a long time, or even recover.

#### SUPPURATIVE INFLAMMATION OF THE PORTAL VEIN.

*Lesions.*—The wall of the vein is thickened and infiltrated with pus. Its cavity is filled with soft, puriform, infectious thrombi. The inflammation and thrombosis extend from the trunk of the portal vein to its branches in the liver. The liver-tissue surrounding the inflamed veins also becomes the seat of suppurative inflammation. Portions of the thrombi may pass into the circulation and lodge as infectious emboli in different parts of the body.

*Causes.*—Fish bones and pieces of wire have been found imbedded in the wall of the vein.

Inflammation of the umbilical vein in infants may extend to the portal vein. Abscesses in the abdominal cavity may be followed by thrombosis and inflammation first of one of the branches of the portal vein and then of the vein itself. Calculi in the common bile-duct, which cause inflammation and ulceration of the wall of the duct, may also cause inflammation of the portal vein. There are cases in which suppurative portal phlebitis occurs without discoverable cause.

*Symptoms.*—The invasion of the symptoms may be sudden, or preceded by a few days of moderate fever, loss of appetite, and general malaise. Pain and tenderness over the liver are constant symptoms. They occur early in the disease and continue throughout its course.



The liver and the spleen are increased in size. Jaundice is often present. There are nausea, vomiting of food and of blood, diarrhœa, and passage of blood from the bowels.

Chills mark the onset of the disease, and are repeated throughout its course. The temperature rises at once and continues through the disease, like the temperature of pyæmia, sometimes high, sometimes low, sometimes below the normal.

There may be ascites, or local or general peritonitis.

The patients rapidly lose flesh and strength, develop cerebral symptoms, and pass into the pyæmic condition. The symptoms are evidently local—dependent on the inflammation of the vein and of the liver; and general—dependent on the septic poisoning.

The disease regularly terminates fatally at the end of about fourteen days; but it may last only a few days, or continue five or six weeks.

#### THE NEW-GROWTHS OF THE LIVER.

Both carcinomata and sarcomata are often developed in the liver as secondary and metastatic tumors. The carcinomata of the stomach, large bile-ducts, and gall-bladder are accompanied with secondary growths in the liver in a large proportion of the cases. It may very well be that the secondary new-growth of the liver attains a large size and gives more marked symptoms than does the primary tumor from which it originates.

Of the primary new-growths of the liver the larger number follow the type of carcinoma. They are composed of tubules lined with cylindrical epithelium, or filled with polygonal cells; or of larger and more irregular spaces packed with polygonal cells.

Primary sarcomata of the liver are of rare occurrence. They follow the type of the sarcomata which seems to be developed from the connective tissue around blood-vessels—angiosarcoma.

The new-growth may take the form of multiple nodules, of single tumors which may reach a large size, or of a diffuse infiltration. The liver itself is usually increased in size, smooth or nodular, according to the arrangement of the new-growth. But some of the livers are not enlarged, some are even diminished in size. The new-growth may compress the bile-ducts, it may compress, or grow into, the veins.

There may be secondary tumors in the peritoneum, ascites, or peritonitis.

The symptoms of the primary new-growth in the liver are variable and often obscure.

1. The patients first complain of derangements of digestion, of gastric dyspepsia, and of functional disturbances of the liver. It often happens that these symptoms follow symptoms of the same kind which existed before the formation of the new-growth in the liver, so that a history of similar symptoms may be given which extends over many years. This means that the patient first suffered from chronic gastritis and functional disturbance of the liver, and then a new-growth in the liver was formed, and the old symptoms were made worse without changing their character. As the tumor of the liver increases in size the patients grow worse. They lose flesh and strength, become anæmic, have an irregular fever, become a little jaundiced, have ascites, œdema of the legs, and a large liver. The progress of the disease is at first slow and gradual, toward the end much more rapid.

2. The patients suffer from the symptoms of chronic gastritis and functional disturbance of the liver. They slowly lose flesh and strength and become anæmic. It becomes more and more evident that they have a malignant new-growth in some part of the body, but it is impossible to locate it on account of the want of local symptoms or physical signs.

3. There are rare cases, with a large nodular liver, which behave like cirrhosis, rather than like cancer.

4. There are cases in which the new-growth very soon invades the branches of the portal veins, so that ascites is an early and prominent symptom before there has been much change in the general health. The ascites may be accompanied by chronic peritonitis. The cases look more like chronic or tubercular peritonitis, or cancer of the peritoneum, than they do like cancer of the liver.

#### CANCER OF THE COMMON BILE-DUCT.

*Lesions.*—The growth begins in the wall of the common bile-duct near the duodenum and surrounds and constricts the duct. The growth may remain small, or may extend to the pancreas and lymphatic glands and form large tumors. The growth constricts the bile-duct, prevents the escape of bile, and so produces dilatation of the bile-ducts and the gall-bladder.

The disease belongs to persons over forty years of age.

*Symptoms.*—Jaundice is developed suddenly and increases in intensity throughout the disease.

Pain comes on suddenly and is referred to the region of the common bile-duct.

Nausea and vomiting often mark the onset of the symptoms and may continue.

The sudden development of the symptoms in a disease of this character is a noticeable feature.

The patients go on with the symptoms of a very bad obstructive jaundice. The pigmentation of the skin becomes constantly deeper, the urine is loaded with bile-pigment, the fæces are white. The patients lose flesh and strength, there are hæmorrhages in the skin and from the mucous membranes, cerebral symptoms are developed, the tongue is brown and dry, the gall-bladder may be dilated so as to form a tumor of some size. The patients finally die deeply jaundiced, emaciated, and semi-comatose.

The duration of the symptoms is usually for from one to three months.

#### CANCER OF THE GALL-BLADDER.

*Lesions.*—The growth is said to begin in the wall of the gall-bladder near the fundus or the cystic duct, and then extends until it involves a considerable part of the bladder. The gall-bladder may become contracted and small, or it may contain gall-stones, or it may be distended with fluid so as to form a tumor of some size. There may be secondary nodules in the liver.

*Symptoms.*—The clinical histories are very obscure. The patients lose flesh and strength and become cachectic, as if they had a cancer in some part of the body.

If the gall-bladder is contracted there is nothing to tell us where the cancer is.

If the gall-bladder is dilated, the presence of the tumor assists in making the diagnosis.

If the liver is enlarged and contains secondary nodules, the case is usually supposed to be one of cancer of the liver.

## BILIARY CALCULI.

The solid portions of the bile may become separated from the fluid portions and form semi-solid, or solid, masses within the biliary passages.

1. Inspissated bile occurs in the form of soft, brown, or greenish masses, composed principally of bile-pigment, with a little cholesterin, the salts of iron, soda, and potash, and sometimes thick, tenacious mucus. While in the biliary passages it retains its viscid consistence, but when dry changes into a fine powder.

2. Biliary calculi are solid bodies composed principally of cholesterin with more or less bile-pigment. They vary as to their size, shape, color, consistence, and number.

*Causes.*—The normal reaction of the bile is neutral or slightly alkaline, if it becomes acid cholesterin is precipitated.

There may be such a large production of cholesterin that it cannot remain in solution.

Any conditions which retard the flow of bile into the intestine, or which render the walls of the bile-passages unhealthy, are liable to produce calculi.

Calculi are rare in children, but of frequent occurrence in persons over twenty years of age. They are more common in women than in men. A sedentary mode of life, constipation, excessive eating of fats, starches, and sugars, and chronic malarial poisoning, seem to have some effect in causing the formation of calculi.

I. CALCULI WHICH ARE FORMED IN THE GALL-BLADDER AND REMAIN THERE.

1. When calculi are formed in the gall-bladder, they may remain there and give absolutely no symptoms. The gall-bladder usually becomes contracted, and the cystic duct is obliterated.

2. Calculi remaining in the gall-bladder may from time to time cause attacks of pain which last for hours or days.

These attacks of pain may be relieved by emetics or by morphine.

In some persons the attacks of pain are repeated frequently, and there is so much loss of flesh and strength that the matter

becomes serious. It is then necessary to open the gall-bladder, and remove the calculi by a surgical operation.

3. Calculi remaining in the gall-bladder may act as irritating bodies, and excite catarrhal or suppurative inflammation of the wall of the gall-bladder.

A catarrhal inflammation is attended with dilatation of the gall-bladder and pain.

A suppurative inflammation is attended with pain, dilatation of the gall-bladder, fever, sweating, loss of flesh and strength. There may be added a localized peritonitis, with the formation of adhesions.

If the patients' general condition remains good, they may be kept in bed, with the application of continuous cold over the gall-bladder. If the cases are at all threatening, the only satisfactory treatment is to open the gall-bladder and remove the calculi.

## II. CALCULI MAY PASS INTO THE CYSTIC DUCT AND BECOME IMPACTED THERE.

1. There will be an attack of biliary colic, or there are several such attacks. After the last attack there will be found a tumor formed by the dilated gall-bladder. This tumor may cause so little inconvenience that no treatment for it is necessary, or it may give so much discomfort that it will be proper to empty it by the aspirator.

2. There will be an attack of biliary colic and, after this, first dilatation and then inflammation of the gall-bladder. The gall-bladder forms a tumor of large size; fever, sweating, loss of flesh and strength are developed; localized peritonitis may be added. If not treated, these symptoms will continue and cause the death of the patient.

Some of these cases may be cured by the continuous application of cold, others by aspiration, while in others it is necessary to open the gall-bladder.

3. Such a calculus may be followed by adhesions between the wall of the bile-duct and the intestine, ulceration, the escape of the calculus into the intestine, and the recovery of the patient. Or the calculus may remain in the intestine and cause an obstruction there.

4. One or more calculi may remain in the cystic duct for years and produce attacks of pain like those due to calculi in the gall-

bladder. These cases have been successfully treated by surgical operations.

### III. CALCULI MAY PASS FROM THE GALL-BLADDER THROUGH THE DUCTS INTO THE INTESTINE.

Such a passage of a calculus is attended with symptoms which are called an attack of biliary colic.

The cases vary as to the size and consistence of the calculi, as to whether they are true calculi or masses of inspissated bile, as to the rapidity of the passage of the calculi, and as to the frequency of the attacks.

*Symptoms.*—Sometimes there is an initial chill, more or less marked ; but this is not constant. Then comes the most marked symptom—pain. This usually comes on suddenly and severely, referred to the epigastrium and the lower edge of the liver, and radiating in different directions. Occasionally the invasion is slower, and the pain more dull and aching. The pain, as such, resembles that of intestinal colic, and that of nephritic colic.

Nausea and vomiting frequently accompany the attack.

Fever occurs in three ways : It may simply accompany the attack. It may be due to a complicating, localized peritonitis. It may be due to pre-existing malarial poisoning.

During an attack of biliary colic, the pulse is usually small and feeble ; the bowels are constipated before and during the attack ; the intestines are distended with gas.

Usually the general condition of the patient remains good, but there may be marked prostration and even collapse and death.

Jaundice is regularly developed within twenty-four hours after the commencement of the pain, and continues for some days after this has subsided. But when the calculi are small, or inspissated bile is passed, the jaundice may be entirely absent. In patients who have repeated attacks of severe pain referred to the upper part of the abdomen without jaundice it is often very difficult to tell whether the pain belongs to the bile-duct, the colon, or the stomach.

The liver may be swollen and tender during and after the paroxysm. The gall-bladder may be swollen, tender, and sometimes surrounded by a localized peritonitis.

*Duration.*—The ordinary attacks only last for a few hours or

days. There may be only one or two attacks, or the attacks are repeated at intervals for a number of years. With such a repetition of the attack the general health may suffer, partly from the recurrence of the pain, partly from the drugs used to control it.

Less frequently there are attacks which last for weeks or months. The pain, jaundice, and vomiting continue, there is an irregular febrile movement, and the patients lose flesh and strength.

*Diagnosis.*—The regular attacks with pain and jaundice are easily recognized.

Repeated attacks of pain in the upper part of the abdomen may be due to the passage of a calculus through the ducts, to the presence of calculi in the ducts or in the gall-bladder, and to disorders of the colon or of the stomach; it is often very difficult to distinguish between them.

*Treatment.*—During the paroxysm of biliary colic the pain is to be relieved by hypodermic injections of morphine, or by inhalations of chloroform or ether. There seems also to be some advantage in the use of hot baths, hot-water enemata, and purgatives.

To prevent the recurrence of attacks of biliary colic there are plans of treatment which are not very rational, but yet which are often efficacious.

In some patients the attacks will cease if the stomach is washed out every day.

In some patients the use of olive-oil stops the attacks. Half a pint can be poured into the stomach through the stomach-tube; or a tablespoonful of oil can be mixed with milk and swallowed, the second day two tablespoonfuls of oil are taken, and so on up to six tablespoonfuls.

The sulphate, chlorate, phosphate, or salicylate of soda seem to cure some cases, as do also the alkaline waters of Carlsbad and Vichy.

If the attacks are frequently repeated and cannot be otherwise relieved a surgical operation is necessary.

IV. CALCULI MAY PASS FROM THE GALL-BLADDER INTO THE COMMON DUCT AND BECOME IMPACTED.

The patients give the history of one or more attacks of biliary colic, or that of several attacks of pain and light-colored fæces without jaundice. Then there comes an attack of biliary colic which lasts for a time and subsides, but the jaundice continues and increases.

There are cases, however, in which there is no attack of pain to mark the time of the lodgement of the calculus. On some particular day it is evident that the common bile-duct is obstructed, and the symptoms of this obstruction become more and more decided.

The skin becomes very yellow, the urine is dark with bile pigment, the fæces are white. There is an irregular fever, the patients lose flesh and strength ; there are loss of appetite, nausea, and vomiting ; the bowels are constipated, the tongue is brown and dry, cerebral symptoms are developed, and there are bleedings from the mucous membranes and into the skin. The liver may be large and tender and the gall-bladder distended.

Unless relieved by a surgical operation this condition regularly proves fatal, but it is possible for the calculus to escape by a perforation into the intestine, and for the patient to recover.

The only treatment is a surgical operation to remove the calculus.

V. CALCULI MAY PASS FROM THE GALL-BLADDER INTO THE COMMON DUCT AND REMAIN THERE WITHOUT BEING IMPACTED.

Such a calculus passes into the common duct and remains there. Then for years the calculus slowly increases in size, and the common and hepatic duct become dilated to make room for it.

The patients suffer from repeated attacks of pain, fever, nausea, vomiting, and jaundice. These attacks become more frequent and the patients cease to improve between the attacks. The jaundice becomes continuous, they lose flesh and strength, and finally die exhausted, or with suppurative inflammation of the gall-bladder or bile-duct.

The only efficient treatment is by a surgical operation.



## VI. CALCULI IN THE INTESTINE.

A calculus of some size, situated in the gall-bladder, may cause inflammation of the wall of the gall-bladder, adhesions to the intestine, perforation, and escape of the calculus into the intestine.

These morbid processes are naturally attended with symptoms, but it sometimes happens that the patient will give no history of them.

After a time the calculus is increased in size by the deposition about it of successive layers of mucus and of the salts of lime.

Such a calculus may remain in the intestine for a long time without any symptoms, except occasional uncomfortable feelings in the abdomen. It may produce attacks of severe pain. It may cause partial or complete obstruction of the intestine.

The only treatment is the removal of the calculus by a surgical operation.







## THE KIDNEYS.

### THE MOVABLE KIDNEY.

It not infrequently happens that one, or sometimes both, kidneys cease to be imbedded in the fat of the lumbar region and become capable of a liberty of motion which increases as time goes on. The kidney continues to be fastened by its large blood-vessels, and pushes the peritoneum in front of it.

It is believed that such a movable condition of the kidney may be either congenital or acquired. It occurs more frequently in women, in women who have borne children, among the laboring classes, and in persons between the ages of twenty-five and forty years. The right kidney is the one most frequently affected, next the left kidney, least often both kidneys.

*Symptoms.*—We can only be certain of the existence of a movable kidney when we are able to distinctly feel it. It feels like a solid tumor, not as hard as the spleen, of the shape of the kidney, movable, easily escaping from the hand which tries to grasp it, easily pushed back into its natural position. In order to make out such a kidney it is necessary to put the patient into different positions, so as to displace the organ ; it is often necessary to examine on several different days before we can be sure of it.

Such a movable condition of the kidney may never cause any discomfort, and the patient may remain entirely unconscious of its existence. In other cases, however, the abnormal position of the kidney gives rise to a variety of symptoms.

Perhaps the most ordinary state of affairs is for the patient either to suffer from pain in the back, or to discover that he has an abdominal tumor.

The pain in the back is referred to the side on which the

kidney is displaced. It is a severe pain, made worse by the erect position. The real cause of the pain is often overlooked for a long time.

The discovery of an abdominal tumor naturally fills the patient with alarm, and so may give rise to a variety of nervous phenomena for which the displacement of the kidney can hardly be held responsible.

Less frequently the variety of symptoms is greater. There are feelings of dragging, of pressure, or of weight in the abdomen. There may be nausea, vomiting, and pain in the abdomen. Intense pains may radiate toward the epigastrium, the sacral and lumbar region, the intercostal spaces, the shoulders, along the ureter, into the testicle or the labia majora.

It is said that there may be attacks of localized peritonitis around the displaced kidney, with chills, fever, intense pain, and hardness and tenderness of the abdomen.

The pelvis of the displaced kidney may become inflamed, or may contain calculi.

By the bending of the ureter there may be produced either temporary or permanent hydronephrosis.

*Treatment* is called for only in those patients in whom the displacement of the kidney gives well-marked symptoms. The simplest plan is to keep the patients in bed on their backs for a month, and then to let them go about with a binder and pad so contrived as to keep the kidney in place.

If the patient cannot be relieved in this way, a surgical operation becomes necessary. The offending kidney may be entirely removed, or it may be fastened in its proper place by sutures.

#### CLASSIFICATION OF DISEASES OF THE KIDNEY.

The recognition of the diseases of the kidney, which still bear the name of their discoverer, dates back only to the year 1827, when Richard Bright published his first paper on the subject. This first paper was followed by others, and in 1829 Christison published in the *Edinburgh Medical and Surgical Review* his account of the same disease. Both these authors regarded the disease as a morbid change in the kidneys, which was the cause of the accompanying symptoms.

In 1841 Rayer completed his large atlas of colored plates

and description of kidney diseases. His classification is as follows :

1. Nephritis—an inflammation of the cortical or tubular portion of the kidneys.

(a) Simple nephritis.

(b) Gouty nephritis.

(c) Rheumatic nephritis.

(d) Nephritis produced by poison.

(e) Albuminous nephritis.

2. Pyelitis.

3. Perinephritis.

In 1842 Rokitansky recognized the waxy, or amyloid, kidneys as presenting different lesions from those found in other examples of kidney disease.

In 1851 Frerichs published his monograph on Bright's disease, and gave a systematic description which has had a decided effect on the minds of most subsequent observers. His conception is that of one disease—Bright's disease, with a characteristic lesion—inflammation of the kidneys. The varieties of the disease depend upon the stages of the inflammation. There are three stages :

1. The stage of hyperæmia and of commencing exudation.

2. The stage of exudation and of commencing transformation of the exudation.

3. The stage of atrophy.

1. *Hyperæmia*.—The first stage is characterized by an increase in the size of the kidneys, especially of the cortex, by general congestion, by extravasation of blood in the Malpighian bodies, the tubes, and the kidney tissue, and by filling of the tubes with coagulated fibrin. The epithelium of the tubes is unaltered.

2. *Exudation*.—In the second stage, the congestion diminishes while the exudation increases. The exudation is found in the tubules and in the interstitial tissue. The exudation between the tubes is sometimes organized into connective tissue. The cortex becomes of a white-yellowish color, and remains thickened. The surface of the kidney is smooth or slightly granular. The pyramids are of a reddish color. Some of the Malpighian bodies are normal, others are enlarged and filled with exudation. In the cortex the epithelium of the tubes is swollen and granular, and may break down altogether, or it simply shrivels and atrophies. The tubes are filled with degenerated epithelium, granu-

lar matter, and fat-globules, or with homogeneous exudation. The tubes are dilated. The dilatation of the tubes is the principal or only cause of the increased size of the kidney.

3. *Atrophy*.—In the third stage, the kidneys are smaller, or of normal size, or even larger than normal. The capsule is adherent. The surface of the kidney is irregular and granular, its color a dusky yellow. Its consistence is hard. The cortex is thinned. The pyramids are smaller. The fat about the pelvis is increased in amount. The tubes are dilated and filled as in the second stage, or are collapsed and folded together. Most of the Malpighian bodies are shrivelled and fatty. If the exudation between the tubes has become organized, we find masses of connective-tissue cells and fibres.

This description of the lesions, taken as it was from nature, is as true now as when it was written. But yet our present pathological knowledge makes us interpret these lesions somewhat differently.

In 1852 Dr. George Johnson published a work on kidney diseases which, like that of Frerichs, has had a durable effect on medical opinions. He distinguishes five forms of Bright's disease.

1. *Acute Desquamative Nephritis*.—The form of disease occurring after scarlet fever, exposure to cold, etc. This corresponds to Frerichs's first stage. Johnson, however, lays most stress upon the desquamation of the epithelium, and but little on the exudation in the tubes. Exudation between the tubes he does not mention.

2. *Chronic Desquamative Nephritis*.—This corresponds to the second and third stages of Frerichs. Johnson describes the degeneration of the epithelium, the denudation of the tubes of their epithelium, their dilatation and collapse, and the presence of coagulated material within them. The Malpighian tufts are thickened or atrophied. The arteries are thickened. He regards the production of new fibrous tissue as an accidental and unessential phenomenon.

3. *Waxy Degeneration of the Kidney*.—Under this name Johnson describes kidneys which are of large size, their cortex thick and white, their tubes filled with waxy material. This waxy material he supposes to be produced by a degeneration of the epithelium. The large hyaline casts found in the urine he calls waxy, and seems to consider them diagnostic of this form of kidney disease.



4. *Acute Non-desquamative Disease of the Kidneys*.—This is characterized during life by scanty or suppressed urine, but containing no albumin, and no casts, or only a few waxy ones. The kidneys are of normal size; the epithelium of the tubes is somewhat altered.

5. *Chronic Non-desquamative Disease*.—The kidneys are usually large, very rarely atrophied. The cortex is thick and white. The convoluted tubes are more opaque than usual. The Malpighian bodies and arteries are thickened.

6. *The Granular Fat Kidney*.—This form may be a consequence of the non-desquamative disease, of acute desquamative inflammation, and rarely of chronic desquamative disease. The kidneys are large, the cortex white, mottled with yellowish granulations. These yellow granulations are formed of tubes containing oil-globules. The vessels and Malpighian bodies are thickened. Sometimes the same yellow, fatty granulations are found in atrophied kidneys.

7. *The Mottled Fat Kidney*.—All the tubes of the cortex contain oil-globules, and there are red spots of congestion or extravasation.

To Traube (1856) belongs the merit of recognizing chronic congestion of the kidney as a lesion with an entirely different cause from that of other forms of Bright's disease; and also of calling attention to the fact that blood-contamination cannot be the only cause of the cerebral symptoms.

In 1858 Virchow, in his Cellular Pathology, developed the doctrine that in Bright's disease either the tubes, the stroma, or the Malpighian bodies are principally involved, and that we can, therefore, distinguish a parenchymatous nephritis, an interstitial nephritis, and an amyloid degeneration of the kidney. This doctrine has had a lasting effect on all subsequent classifications.

Grainger Stewart distinguishes:

1. *The Inflammatory Form*.—This has three stages: (1) That of inflammation; (2) that of fatty transformation; (3) that of atrophy. These correspond very closely with the three stages described by Frerichs.

2. *The Waxy Form*.—This also has three stages: (1) That of simple degeneration of the vessels; (2) that in which a secondary alteration of the tubes is superadded; (3) that of atrophy.

In the first stage, the kidney is of normal size, the tubes are

unaltered ; only the Malpighian bodies and small arteries have undergone waxy degeneration.

In the second stage the kidney is enlarged, the cortex thick and white, with Malpighian bodies and small vessels waxy ; the tubes contain hyaline casts ; their epithelium is swollen ; their basement membrane may be waxy.

In the third stage the kidney is small. The surface is rough, granular, and pale. The tubular structures are swollen. The tufts and vessels are waxy. A few tubes are distended, most are collapsed, and are represented only by fibrous tissues.

3. *The Cirrhotic, or Contracting Form.*—This consists of an hypertrophy of the connective tissue of the organ, and a consequent atrophy of all the other structures.

There is at first little diminution in the size of the organ, but the capsule is thickened and adherent, and the surface is rough and granular. The color is pale and reddish. The arteries are prominent, their walls thickened, and their cavities often dilated. On the surface, and in the substance, cysts are often seen. Some are produced by dilatation of the Malpighian capsules, some by dilatation of the tubes, some by a morbid growth of epithelial elements. The tubes are compressed and atrophied by the new fibrous tissue. They contain little opaque material, but often hyaline matter. Sometimes urate of soda is found in the stroma and tubes of the pyramids. The disease is a non-inflammatory increase of connective tissue.

Both the waxy and contracting forms may be secondarily affected with the inflammatory disease.

4. *Simple Fatty Degeneration.*—The kidneys are of about the normal size. The surface is smooth, the capsule not adherent. Their texture is soft, the cortex is pale and mottled, with sebaceous-looking deposits. The epithelium of the tubes is fatty.

Dickinson describes tubal nephritis, granular degeneration, and depurative infiltration :

1. *Acute Tubal Nephritis.*—This, the nephritis of scarlet fever and of exposure to cold, is described in very much the same terms as the acute desquamative nephritis of Johnson.

2. *Chronic Tubal Nephritis.*—The kidney is large, the cortex of an opaque white or buff color, the pyramids pink. The surface is smooth, the capsule not adherent. The convoluted tubes are distended with granular and fatty epithelium and with fibrinous exudation. The straight tubes are packed with the products of

epithelial growth, while others contain transparent fibrin. The tubes are not changed, save as regards their contents. The-Malpighian bodies are normal or somewhat dilated. There is no increase of inter-tubular tissue. These kidneys remain large and smooth to the last, unless complicated with the depurative change.

Sometimes the cortex is sprinkled with white, sharply defined specks, like bits of bran. This change is characteristic of a great amount of fatty change in the accumulated epithelium.

3. *Granular Degeneration*.—The kidneys may be of normal or even increased size, but are usually small. The capsule is adherent. The surface is irregular and covered with little rounded nodules. The cortex is thin. Cysts are often found in the cortex and cones. There is an increase of fibrous tissue around the Malpighian bodies and vessels, and beneath the capsule and deeper in the cortex. The cortical tubes are atrophied or dilated, but many tubes may remain unchanged. The tubes may be filled with epithelium, or with transparent, fibrinous material. In the majority of cases the epithelium is exactly such as is found in normal kidneys. When changed, it is by an alteration in its regularity of form, becoming somewhat angular, as if cramped in growing space. The circulation through the blood-vessels is much obstructed. The formation of cysts is due to dilatation of the tubes or of the Malpighian capsules.

4. *Depurative Infiltration*.—The kidney is at first of normal size, pale, and its surface smooth. The only change is in the Malpighian tufts, which react with iodine. As the disease goes on, the kidney becomes larger and its capsule adherent. The cortex is of a pale, opaque fawn-color, or has a pinkish or gray translucency. Afterward the kidney atrophies and its surface becomes nodulated. There may be small cysts. In cases of long standing, almost the entire organ gives the characteristic reaction with iodine. The first change is the infiltration of the Malpighian bodies and vessels. Afterward new fibrous tissue is formed between the tubes, the epithelium degenerates, the tubes are dilated and contain fibrinous casts.

It will be seen that the name of "depurative infiltration" is given to the same form of kidney disease which is called by others waxy or amyloid.

Klebs describes :

1. *Diffuse Granular Degeneration of the Epithelium*.—This con-

dition is found by itself, and in connection with lesions in the interstitial tissue. By itself, it occurs with pyæmia, phthisis, rheumatism, typhoid and typhus fevers, the malarial fevers, the acute exanthemata, extensive burns, poisoning with phosphorus and the mineral acids. During life the urine may contain granular casts and albumin. The kidney is somewhat enlarged, the cortex grayish-yellow, the pyramids bluish-red. There may be little extravasations of blood in the convoluted tubes. The epithelium of the tubes is granular and may distend them. The tubes may contain casts. These changes are most frequent in the convoluted tubes, but are sometimes confined to the straight tubes of the pyramids. The entire process is a degenerative and not an inflammatory one.

2. *Cyanotic Induration of the Kidneys*.—This condition is produced by any long-continued obstruction to the escape of venous blood from the kidneys, most frequently by heart disease. The kidneys are increased in size, the surface smooth, the capsule not adherent. The organ is hard, the cortex and pyramids congested and of dark-red color. The epithelium of the tubes is not altered. The interstitial tissue is harder, but not increased in amount. The continued congestion may, after a time, produce further changes. The epithelium of the convoluted tubes may undergo granular degeneration, and the cortex becomes paler. Or there may be an increase of interstitial tissue, and the surface becomes nodular.

3. *Interstitial Nephritis*.—This has two stages : (a) That of cell-infiltration ; (b) that of atrophy.

(a) *The Stage of Cellular Infiltration of the Interstitial Connective Tissue*.—The kidney is increased in size. The surface is smooth, the capsule not adherent. The cortex is of a whitish or yellowish color, the pyramids red. In the cortex the tissue between the tubes is everywhere increased from two to four fold. This increase is due to the presence of lymphatic elements and of clear serum. There is at first an exudation of lymphatic fluid, which dilates the lymphatic vessels of the interstitial tissue, and is accompanied by an emigration of white blood-globules, which finally fill all the spaces in the interstitial tissue. The epithelium of the convoluted tubes undergoes granular degeneration in consequence of its disturbed nutrition. The increased pressure of blood causes an exudation of the elements of the blood from the Malpighian tufts, namely, fibrinogenic material which coagu-

lates in the tubes, albumin, and red blood-globules. The lymphatic cells perforate the basement membrane of the tubes and become adherent to the fibrinous cysts.

(b) The Stage of Atrophy.—The preceding stage may terminate in resolution and recovery. If it does not, it is succeeded either by a hyperplasia of connective tissue or by granular atrophy.

If there is a hyperplasia of connective tissue, the kidneys are of normal size, or slightly atrophied. The capsule is somewhat adherent. The cortex is whitish, yellowish, or mottled. The pyramids are congested. There is a uniform increase of connective tissue between the tubes. The tubes are unaltered or somewhat narrowed.

Granular atrophy is more common. The kidney is atrophied. The capsule is very adherent. The surface is uneven and nodular. The change of the lymphatic cells into connective tissue is accompanied by fatty degeneration of the cells. In the atrophied spots the tubes and glomeruli become impervious. The tubes contain hyaline casts. The basement membrane of the atrophied tubes becomes thick and fibrous. The glomeruli are atrophied, their capsules thickened, their vessels obliterated. The larger arteries are thickened.

Glomerulo-nephritis.—Klebs gives this name to a form of disease which he has observed in scarlatina cases. The kidneys are of medium size, the capsule not adherent, the surface smooth, the parenchyma congested. There are no changes except in the glomeruli. These appear as opaque, white points. On minute examination, it is found that there are large numbers of small, rounded cells about the loops of the Malpighian tuft, while the epithelium of the capsule is unaltered.

Amyloid degeneration is described in much the same way as by other authors.

Rindfleisch describes :

1. *Acute Parenchymatous Nephritis*.—In the milder form the kidney is of normal size, the surface smooth, the cortex of a yellowish-gray color. There is a moderate degree of cloudy swelling of the epithelium of the convoluted tubes.

In the severer form the kidney has the same appearance, but is increased in size and the cortex is thickened.

Both these forms occur with the acute exanthemata, typhus fever, pyæmia, etc.

2. *Diffuse Interstitial Nephritis*.—This corresponds very closely with the description given by Klebs.

He states that the disease may begin as a parenchymatous nephritis, and afterward become interstitial, but that the two forms also occur independently of each other.

*Amyloid degeneration* is usually accompanied by interstitial nephritis. The amyloid degeneration is the primary change, and the nephritis follows it as a secondary lesion.

Rosenstein describes :

1. *Chronic Congestion of the Kidney*.—This condition is described in much the same way as by the preceding authors.

2. *Catarrhal Nephritis*.—The kidney is of normal size, or slightly enlarged; in severe cases congested and mottled with small ecchymoses. The process begins at the apices of the pyramids, which are at first congested, afterward pale. After a time we find the pyramids divided into red and white striæ, running from the apex to the base of the pyramids. The red striæ are the portions more recently congested; the white are the tubes distended by an increase of epithelium.

The urine contains a little albumin, hyaline, granular, and epithelial casts and blood-globules.

The symptoms during life are not marked. The lesion is seldom primary. It may follow catarrhal inflammation of the urethra, bladder, or ureters; the use of cantharides, copaiba, or cubebs, typhoid or typhus fever, cholera, etc.

3. *Diffuse Nephritis, Parenchymatous Nephritis, Bright's Disease, Granular Degeneration of the Kidney*.—This form has three stages.

The first stage is that of hyperæmia. The kidney is of normal size, or enlarged, congested, and red; there is blood in the tubes, and the epithelium of the convoluted tubes is swollen.

The second stage is that of exudation. The kidney is enlarged, the cortex pale, the pyramids red. The epithelium of the convoluted tubes is swollen and granular. The tubes are dilated and contain casts. There is usually an increase of cells in the interstitial tissue.

The third stage is that of atrophy. The kidney becomes smaller, its surface nodular. The atrophy may take place without any change in the interstitial tissue, simply as a result of the destruction of the epithelium. Usually, however, the retraction of the new interstitial tissue assists in producing the atrophy.

The epithelium is granular or fatty. The Malpighian bodies

are atrophied, their capsules thickened and surrounded with new connective tissue. The basement membranes of the tubes are thickened, and are accompanied by bands of connective tissue. The intertubular capillaries are partly dilated, partly small and fatty.

The atrophy consists, therefore, in a suppression of the function of a number of the tubes, with obliteration of some of the blood-vessels and increase of the interstitial tissue.

Either process, that in the epithelium or that in the connective tissue, can occur separately, but they are usually combined.

4. *Amyloid Degeneration*.—Rosenstein describes this form in much the same way as other authors. He regards the degeneration of the vessels only as a complication of the parenchymatous and interstitial change.

5. *The Fatty Kidney*.—There is an infiltration of the epithelium with fat, or a fatty degeneration. The condition is described in the same way as the diffuse granular degeneration of Klebs.

Weigert divides Bright's disease into parenchymatous degeneration and true nephritis. He does not distinguish between interstitial and parenchymatous forms of nephritis, but believes that in all cases the disease begins with degeneration of the epithelium, which is followed by inflammatory interstitial processes.

Gull and Sutton have shown very clearly the frequency with which changes in the arteries and capillaries—arterio-capillary fibrosis—are associated with the atrophic form of Bright's disease; and that these changes in the arteries and capillaries may also exist and give symptoms without any lesions of the kidneys. From these facts they have drawn the conclusion that this form of Bright's disease is not, properly speaking, a disease of the kidneys, but rather one of the arteries and capillaries.

Bartels uses the name of "The Diffuse Diseases of the Kidneys," with the subdivisions of Hyperæmia, Ischæmia, Acute Parenchymatous Nephritis, Chronic Parenchymatous Nephritis, Renal Cirrhosis, and Amyloid Degeneration.

*Active hyperæmia* is, he says, a condition which arises slowly as the result of some toxic influence, most frequently from the use of cantharides. Apparently he has little or no personal experience of the anatomical changes found in the kidneys.

*Passive hyperæmia* is the same condition as that also called "chronic congestion of the kidney," and cyanotic induration of the kidney. Its most important form is that due to valvular

lesions of the heart and to certain affections of the lungs. He makes no new statement concerning the pathological changes.

*Ischæmia* is the condition of more or less complete stoppage of the arterial blood-supply to the kidneys, occurring independently of congestion of the nervous system. It occurs only in the asphyxia stage of cholera.

*Acute Parenchymatous Nephritis.*—Under this name Bartels includes all the cases of acute Bright's disease. He says that the only essential and constant microscopical appearances are the changes in the epithelium of the tubes. The epithelial cells are swollen and cloudy, they are infiltrated with granules of fat, and are broken-down. To these changes in the epithelium are frequently added a swelling and infiltration of the stroma, casts in the tubes, and extravasations of blood.

*Chronic Parenchymatous Nephritis.*—This may follow acute parenchymatous inflammation, or may begin as a chronic process. The kidneys are large, white, and smooth. The tubes are dilated; the epithelium is only partly preserved, and the cells which remain are large, granular, and fatty. In many places the epithelium is completely gone and in its stead the tubes are entirely filled with masses of detritus mixed with oil-globules; casts are found in many of the tubes. The stroma is thickened by fluid exudation, by an emigration of white blood-cells, and by a growth of new connective tissue. The small arteries and Malpighian tufts are often the seat of waxy degeneration.

*Renal Cirrhosis.*—The kidney is very much diminished in size, especially the cortex. This diminution in size is due to the wasting of the glandular tissue, while at the same time there is an extensive growth of new fibrous tissue. The change in the kidney is due to a primary growth of the intertubular connective tissue, and this leads to the dwindling of the substance of the gland, a wasting preceded by no inflammatory swelling of the organ.

To Cohnheim belongs the merit of drawing attention to the importance of the glomeruli in acute nephritis, and to the changes which are found in them. He also points out clearly that well-marked symptoms of acute nephritis may exist during life, although no structural changes are found in the kidneys after death.

Langhans, Nauwerck, and Friedländler have developed still further the doctrine of glomerulo-nephritis, and have described in detail the lesions found in the glomeruli.



Ziegler describes :

1. *Glomerulo-nephritis*, occurring either by itself or combined with changes in the epithelium of the tubes, or with exudation of inflammatory products into the stroma.

2. *Chronic Parenchymatous Nephritis*.—The common feature of this form of nephritis is that there is a continuous inflammatory exudation from the blood-vessels, and that changes go on in the epithelium of the kidney.

As subdivisions of parenchymatous nephritis he distinguishes :

The inflammatory, fatty kidney ;

Chronic hemorrhagic nephritis ;

Chronic glomerulo-nephritis.

3. *Chronic Indurative Nephritis*.—The inflammation leads to a new growth of connective tissue in the stroma, and an atrophy of the tubes and the glomeruli.

4. *The Arterio-sclerotic Contracted Kidney*.—In this the changes begin in the walls of the arteries ; they are thickened, their lumen is narrowed or obliterated. As a result, smaller or larger numbers of glomeruli become atrophied, with the kidney tissue belonging to them. The stroma is not much thickened.

Cornil remarks that the expression of Bright's disease applied to the kidney has nowadays no more value than the expression of dyspepsia applied to the pathology of the stomach or of asystolie applied to the pathology of the heart. He distinguishes :

1. *Granular alteration of the renal epithelium*—a common lesion found in different forms of hyperæmia, especially in those symptomatic of the infectious diseases.

2. *Fatty degeneration*, which is secondary to the chronic diseases.

3. *Chronic congestion of the kidney*, due to mechanical disturbances of the circulation.

4. *Diffuse nephritis*, in which all the anatomical elements of the kidney are involved.

(a) Acute nephritis with predominance of congestive and inflammatory phenomena.

(b) Acute nephritis with predominance of diapedesis.

(c) Acute nephritis with predominance of degenerative lesions.

(d) Nephritis with predominance of the lesions of the glomeruli.

(e) Nephritis with predominance of lesions of the epithelium.

(f) Nephritis with predominance of lesions of the stroma.

5. *Systematic nephritis*, in which from the first the lesion involves exclusively one of the elements of the kidney.

(a) Epithelial cirrhosis of the kidney. A chronic degeneration of the renal epithelium with atrophy of certain systems of tubes and glomeruli.

(b) Vascular cirrhosis—a true interstitial inflammation of the kidney with endarteritis of the renal arteries.

All of Cornil's anatomical descriptions are very true to nature, but his classification is not one adapted to clinical purposes.

As we look back over the history of the disease, it is easy to recognize the points of difference and the progress which has been made.

From the very first we find authors looking at the disease from two points of view: that of the symptoms and that of the lesions. So that, while some regard Bright's disease as a nephritis with its attendant symptoms, others regard it as a disease of the blood, or of the arteries and capillaries, with which a nephritis may, or may not, be associated.

At the time when Frerichs wrote, it was customary to regard a great many morbid conditions as of an inflammatory character, and to think that every inflammation went regularly through three stages. So we find Frerichs arranging all the lesions of Bright's disease as belonging to the stages of congestion, exudation, and contraction of a nephritis, and teaching that all the forms of acute and chronic Bright's disease were different stages of one and the same morbid process.

Then we find in England, first Johnson, and then Dickinson, referring most of the kidney lesions to changes in the epithelium of the tubes. Here, again, it soon became evident, that although changes in the epithelium exist and are of importance, yet Johnson and his school had taken too one-sided a view of the subject.

That in some cases of Bright's disease there is waxy degeneration of the walls of the arteries and Malpighian tufts was early recognized by Rokitansky. As these kidneys have been more studied, it has been found that there may be:

1. Waxy degeneration of the arteries and glomeruli, without any change in the other parts of the kidneys or any disturbance of its functions.

2. Waxy degeneration of the arteries and tufts, followed by chronic changes in the rest of the kidneys.

3. Waxy degeneration of the arteries and tufts, forming an unimportant part of a chronic nephritis.

The next step forward was the recognition by Traube of the condition of chronic congestion of the kidney, its dependence on heart disease, and its termination in changes in the structure of the kidney.

Then we find an attempt by Grainger Stewart to go back to Frerichs's classification of an inflammation in three stages, but separating the waxy and the cirrhotic kidneys.

Among English writers we find a disposition to class the kidneys according to their gross appearance, and to speak of the large white kidney and the contracted kidney, and to regard the cirrhotic kidney as not inflammatory. In England, also, we find especial attention drawn to the condition of the arteries and capillaries in the kidneys and in the rest of the body as a cause of the kidney lesions and of the symptoms.

The next step forward was the recognition of the changes in the glomeruli, first by Klebs, then by Cohnheim, Friedländer, and others.

At the same time there has been an attempt, especially in Germany, to class together the changes in the epithelium, the exudation of inflammatory products, and the formation of new connective tissue under the one head of parenchymatous inflammation, teaching that the morbid process originates in the epithelium, and that the other changes are secondary to this.

Further modifications have been introduced into this doctrine of parenchymatous nephritis, by the contention that changes in the epithelium alone are not of inflammatory, but of degenerative, nature. Incidentally Cohnheim brings out well the important point that, with well-marked changes in the urine and constitutional symptoms we may find no structural changes in the kidneys; in other words, that the morbid changes in these kidneys must have been confined to the blood-vessels.

With Ziegler, Cornil, and others have come in an improved technique and an exact study of the changes in the kidney, which have given us a much more satisfactory knowledge of the lesions.

Although so much has been done in the study of the lesions of Bright's disease, it must be confessed that the ideas of the profession in general concerning it are still somewhat crude.

As regards acute Bright's disease, we often find the belief :

That the kidneys are large, and either white or congested ; that the chief change is in the epithelium of the tubes, which is swollen and detached and blocks up the tubes ; that there is some change in the glomeruli which allows albumin to pass through the walls of the capillaries ; that the patients pass too little urine ; that in consequence of this diminished production of urine there may be developed dropsy or cerebral symptoms ; that the chief object of treatment is to make them pass more urine, or, failing this, to purge or sweat them.

As regards chronic Bright's disease, it is generally believed that there are two principal forms : one, in which the kidney is more or less large and white, while during life there is dropsy and much albumin in the urine ; and one, in which the kidney is more or less contracted and red, and there is little or no albumin in the urine, and little or no dropsy.

It has become evident to many careful observers that there is a group of persons who are more liable than are others to chronic productive inflammation in different parts of the body. It may be the lungs, or the heart, or the arteries, or the liver, or the kidneys that are affected ; and either one or several of these organs are involved at the same time.

The liability is most common after forty-five years of age, but is by no mean infrequent in younger persons. Unquestionably many of these persons are gouty ; in some there is a history of chronic alcoholism ; in some there is an hereditary history ; many of them suffer from disturbances of digestion, many of them have habitually an excess of urates, or oxalates, or occasional sugar, or albumin in the urine.

So great is the number of these cases, and so constantly are these persons under observation, that it is often not difficult to recognize that an individual belongs to this group before he has developed any one of the characteristic inflammations. We can predict beforehand that a given individual will, at some time, develop emphysema, or chronic endocarditis, or endarteritis, or cirrhosis of the liver, or chronic nephritis.

Curiously enough, it has occurred to some very intelligent physicians that this group of persons are really all suffering from the same disease, and that they develop the characteristic lesions as the result of the disease. They propose to call this disease Bright's disease. According to this view a person can have

Bright's disease while the kidneys are still normal. In this way have come in the terms of "renal inadequacy" and of the "pre-albuminuric stage of Bright's disease."

There can be no question that this group of cases is a very important one, and it is quite true that most of them do have disease of the kidneys before they die. I do not know of any good name to designate all the cases of this kind, but I do not think we have any right to say that they are all examples of one disease. Much more probable is it that they all exhibit the effects of heredity, environment, and mode of life.

There is a well-marked disposition on the part of some authors to include cases of chronic inflammation of the arteries under the name of Bright's disease. This view of the matter is clearly stated by Mahomed in Guy's Hospital reports for 1880.

He says: "The object of this paper is to prove that in the earlier stages, and in most cases even to their final stage, the urine of what is generally known as chronic Bright's disease with red granular kidney is most commonly perfectly normal. More than this, its object is to prove, either that chronic Bright's disease is not a renal disease, although it frequently gives rise to a renal affection, or else that another disease must be recognized which constantly precedes and prepares the way for Bright's disease, which may be called arterio-capillary fibrosis, or any other name that may be preferred to it."

The cases which Mahomed narrates in his paper seem to be cases of chronic arteritis, with more or less complicating nephritis.

In the present state of our knowledge it is wiser to put aside the name of Bright's disease and the ideas connected with it, and look for a classification of kidney diseases which will be of practical clinical use and anatomically correct. There seem to be three ways in which we can classify kidney diseases: according to their causes, according to the part of the kidney involved, or according to the nature of the morbid process.

To classify kidney diseases according to their causes is, in the present state of our knowledge, simply impossible. If, for example, we try to make a class of the kidney diseases caused by scarlet fever, we find that the poison of this disease produces three kidney lesions which differ from each other clinically and anatomically. On the other hand, one well-marked form of acute nephritis is caused by scarlet fever, by diphtheria, by pregnancy,

and occurs without discoverable cause. That all forms of nephritis are caused by irritating substances in the blood is very probable; that different quantities of the same poison can produce different forms of inflammation has been demonstrated, but we are still very far from being able to construct a classification based on causes.

To classify kidney diseases according to the part of the kidney principally involved is very natural and not at all difficult. There can be no question that disease of the epithelium, of the glomeruli, of the stroma, or of the arteries decidedly predominates in different sets of kidneys. A classification on this basis is anatomically correct. But when we try to use this classification for clinical purposes it does not answer. The history which I have already given of anatomical classifications shows this only too plainly. A classification according to the nature of the morbid process is altogether the most promising. There are three morbid processes which occur in nearly every part of the body which produce definite anatomical changes, cause regular clinical symptoms, and call for appropriate methods of treatment. These morbid processes are congestion, degeneration, and inflammation.

*Congestion*, whether acute or chronic, produces an accumulation of blood in the veins and capillaries of the part affected, causes local symptoms and disturbances of function, and is to be relieved by means addressed to the circulation of the blood.

*Degeneration*, whether acute or chronic, produces changes more or less profound in the parts affected, is regularly caused by disturbances of circulation, and by other diseases; produces disturbances of function according to its severity; may be itself a cause of inflammation, and can be but little affected by treatment.

*Inflammation* is attended with three essential features, which may occur separately or together: an escape of the elements of the blood from the vessels, a formation of new tissue, and a death of tissue. So we speak of exudative, productive, and necrotic inflammations.

(a) Exudative inflammation is of short duration, leaves behind it no permanent changes in the parts affected, is sometimes accompanied by the growths of pathogenic bacteria, and can be favorably affected by treatment.

(b) Productive inflammation runs an acute, subacute, or

chronic course. It effects permanent changes in the inflamed parts. Its acute forms are very apt to become chronic. There is much variety as to the relative quantity of exudation and of new tissue. Pathogenic micro-organisms may be present.

(c) Necrotic inflammation is characterized by the addition of death of tissue to an inflammation of either exudative or productive type. It is always accompanied by the growth of pathogenic bacteria.

Applying this principle of classification to the kidneys we can distinguish :

1. Acute congestion of the kidney.
2. Chronic congestion of the kidney.
3. Acute degeneration of the kidney.
4. Chronic degeneration of the kidney.
5. Acute exudative nephritis.
6. Acute productive nephritis.
7. Chronic nephritis with exudation.
8. Chronic nephritis without exudation.
9. Suppurative nephritis.
10. Tubercular nephritis.

## THE URINE.

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### QUANTITY.

In healthy adults consuming the ordinary quantities of fluids and solids the daily discharge of urine is 1,250 c.c., or 50 fluid ounces, or 3 pints. This quantity is liable to a considerable amount of variation according to the quantity of fluid taken and the amount of perspiration.

Complete occlusion of the pelves of the kidneys or of their ureters leads to complete suppression of urine. It is not that urine is formed and cannot escape on account of the occlusions, but that the kidneys cease to perform their functions. Suppression of urine is always fatal, but yet can be borne for a number of days almost without symptoms. Sooner or later, however, prostration, delirium, stupor, and the typhoid state are developed.

Severe injuries and surgical operations, especially those on the urethra and bladder, may be followed by a suppression of urine which is often fatal. It is probable that this suppression is due to an acute congestion of the kidneys. Any disturbance of the circulation which produces either acute or chronic congestion of the kidneys regularly diminishes the quantity of urine. So we find that in acute and chronic congestion of the kidney, in acute nephritis, in the exacerbations of chronic nephritis, and in attacks of contraction of the arteries the quantity of urine is notably diminished.

When the body temperature is considerably higher than the normal the urine is diminished in quantity.

In saccharine diabetes patients pass very large quantities of urine, the kidneys being apparently excited to increased activity by changes in the composition of the blood. In cases of insipid diabetes the quantity of urine of low specific gravity is large, but it is not determined why this increase takes place.

In the slow forms of chronic nephritis, whether with or with-



out exudation, it is the rule to have increased quantities of urine of low specific gravity, the quantity being especially large and the specific gravity very low if the vessels are the seat of waxy degeneration. But this increased production of urine may at any time be checked by changes in the heart's action, by contraction of the arteries, or by an exacerbation of the nephritis.

#### SPECIFIC GRAVITY.

The determination of the specific gravity of the urine gives us the relative quantity of its solid and fluid constituents. To obtain practical information on this point it is necessary to examine the urine passed at different times in the day on a number of days. In healthy persons and under ordinary conditions, the specific gravity ought not to vary much from 1020. It seems to be generally agreed that the solid portions of the urine are excreted by the epithelium of the convoluted tubes and the fluid portions filtered through the Malpighian bodies. We would expect, therefore, that a diminution in the specific gravity would be caused by changes in the renal epithelium, and a diminution in the quantity of the urine by atrophy of the Malpighian bodies. As a matter of fact, the kidneys behave differently. When the morbid changes are confined to the epithelium, as in acute and chronic degeneration, the specific gravity is not lowered; when the Malpighian bodies are atrophied in chronic nephritis the quantity of urine is not necessarily diminished. A persistent low specific gravity means a chronic nephritis with a large production of new interstitial connective tissue, or with waxy degeneration of the blood-vessels; or it means insipid diabetes. In chronic nephritis the specific gravity remains low even if the quantity of urine is very much diminished. But in insipid diabetes the specific gravity rises as the urine is diminished in quantity.

An increase in the specific gravity regularly accompanies saccharine diabetes and chronic congestion of the kidneys.

#### CONSTITUENTS, NORMAL AND ADVENTITIOUS.

*Urea*.—The most important of the solid constituents of the urine is *urea*, of which a healthy adult excretes every day about 500 grains. While the specific gravity of the urine gives a gen-

eral idea of the relative quantity of urea, yet there are sources of error. It is best to get the whole daily excretion of urea for several days by the hypobromite method. The principal importance of this is in determining the prognosis of cases of chronic nephritis. When the daily excretion of urea is much below the normal the prognosis is bad, although the patients may seem to be doing well.

*Urates, Oxalates, and Phosphates.*—The presence of an excess of uric acid, of the urates, of oxalate of lime, and of the phosphates are of importance, not because they indicate disease or disturbance of functions of the kidneys, but because they show disordered digestion and an abnormal condition of the blood. There are many cases of kidney disease in which the treatment of these disturbances is of the greatest importance.

*Blood.*—Hæmaturia is an evidence of bleeding from some part of the genito-urinary tract. So far as the kidneys are concerned the blood comes from their pelves, or from the kidneys themselves. Bleeding from the pelves occurs with pyelitis, with calculi in the pelvis, and with new growths of the pelvis. Bleeding from the kidney itself is found with acute nephritis, with exacerbations of chronic nephritis, with tubercular nephritis, with the hemorrhagic forms of the infectious diseases, and with malignant growths.

*Hæmoglobinuria.*—There are morbid conditions in which a considerable number of red blood-cells are suddenly killed and the coloring matter set free in the blood. This is followed by a discharge of this coloring matter, with a considerable transudation of blood serum from the kidneys, in the urine. We find then a good deal of albumin and of red coloring matter in the urine, but no red blood-cells.

*Casts.*—There has been some difference of opinion as to the mode of formation of the little cylindrical bodies which are found in the urine and in the kidney tubules. The question has been whether they are all formed of substances coagulated from the blood plasma, or whether some are formed of substances derived from the renal epithelium. Certainly most of them are formed from the blood plasma. They are composed of a transparent, homogeneous matter with which may be mixed renal epithelium, white and red blood-cells, and the granular matter, fat, and nuclei derived from degenerated epithelium. The presence of casts in the urine means, therefore, that there has been

an exudation of blood serum into the kidney tubules and more or less degeneration of the renal epithelium. The number of casts in the urine is usually an indication of the number formed in the kidneys, but not always; we may find but few casts in the urine during life, and yet after death the kidneys are seen to contain a large number. Albumin and casts are usually present in proportionate quantities: if there is much albumin there are generally many casts, but albumin may be present in large quantities with very few casts. The centrifugal machines which are now in use are of great assistance in looking for casts.

Anyone who wishes to understand casts and their mode of formation must look at them in kidney sections, as well as in the urine. It seems hardly necessary to warn against confounding cylindrical strings of mucus formed in the bladder, often having crystals imbedded in them, with casts formed in the kidney tubules, but the mistake is sometimes made.

Hyaline casts in small numbers, like albumin in small quantities, are occasionally present without disease of the kidneys.

Acute congestion of the kidneys often gives hyaline casts, sometimes granular and nucleate casts.

Chronic congestion gives a few hyaline casts.

Acute degeneration gives casts according to its severity—hyaline casts only, or granular and nucleated casts, or epithelial and blood casts.

Chronic degeneration gives only a few hyaline casts, or none at all.

Acute exudative and acute diffuse nephritis give many casts of every kind.

Chronic nephritis with exudation gives many casts of all kinds, their number much increased when there is an exacerbation of the nephritis.

Chronic nephritis without exudation gives a few hyaline casts or none at all.

### DROPSY.

The association of dropsy with kidney disease is of such frequent occurrence that it is often difficult to convince both patients and physicians that "Bright's disease" can exist when dropsy is absent.

If we go through the list of diseases of the kidney we find that their association with dropsy is as follows :

*Acute congestion of the kidney*—no dropsy.

*Chronic congestion of the kidney*—dropsy according to the condition of the heart.

*Acute and chronic degeneration of the kidney*—no dropsy.

*Acute exudative nephritis*—subcutaneous dropsy, most frequent with nephritis caused by scarlet fever, or by exposure to cold. A similar subcutaneous œdema can be produced by scarlet fever or by exposure to cold without nephritis.

*Acute productive (or diffuse) nephritis*—both subcutaneous dropsy and dropsy of the serous cavities.

*Chronic nephritis with exudation*—dropsy of the subcutaneous connective tissue and of the serous cavities in nearly every case.

*Chronic nephritis without exudation*—no dropsy until late in the disease unless from complicating lesions.

*Suppression of urine* from obstruction of the ureters—no dropsy.

The primitive explanation of renal dropsy was that fluids accumulated in the body because the patient passed too little water. This seemed satisfactory even to so good an observer as Bartels. It has always had its effect on therapeutics; the rule has been, if a patient has dropsy make him pass more urine. This explanation, however, is in constant contradiction with clinical experience.

The present condition of our knowledge on this subject may be stated somewhat as follows :

1. All dropsies are due to an increased transudation of blood serum from the capillaries and a diminished absorption by the lymphatics. The increased transudation is the more important part of the process.

2. Inflammatory dropsies (or exudations) and passive dropsies may be produced in one of two ways :

(a) The capillaries act as filters. Either increased pressure, a change in the composition of the serum, or a change in the walls of the capillaries can cause an increased transudation through the walls of the capillaries.

(b) The capillaries with their endothelium act as glands and secrete serum. Changes in the composition of the blood or irritating substances in the surrounding tissues can irritate the endothelium and cause increased secretion.

Following these rules, the probable explanations of renal dropsies are :

1. *In acute exudative nephritis* the dropsy is due to inflammatory changes in the skin. The dropsy is regularly confined to the subcutaneous connective tissue, and is especially frequent when the nephritis is caused by scarlet fever, or by exposure to cold.

2. *In acute productive nephritis* and in chronic nephritis with exudation the dropsy involves both the subcutaneous connective tissue and the serous cavities. It may be due to irritating substances in the blood, to changes in the composition of the blood, or to changes in blood-pressure.

3. *In chronic nephritis* without exudation, the dropsy which comes on late in the disease is caused by changes in blood-pressure due to heart failure.

Scattered through medical literature are reports of cases of general subcutaneous dropsy coming on suddenly, lasting for a short time, without any evidence of renal or other disease, and terminating in recovery. Traube thinks that such dropsies are due to a disturbance of the functions of the skin caused by exposure to the weather, but this explanation will not answer for all the cases. In some patients (Taylor, *Medical Times and Gazette*, 1871) the dropsy was preceded by a well-marked febrile movement. I only know of one case of this kind which terminated fatally. It is reported by Wernicke (*Deutsch. Archiv für klinische Medicin*, VI., 622). The patient, a woman twenty-two years old, died apparently from the dropsy, and the autopsy showed no lesion to account for the symptoms. The following case is an example of this form of dropsy :

Male, 26, admitted to the Roosevelt Hospital on April 17, 1884. He had been perfectly well and working hard two months ago. Then he began to have cough, mucous sputa, and wheezing breathing. One month ago he developed general subcutaneous œdema; the urine was somewhat diminished in quantity, but the man did not feel sick. When admitted to the hospital his pulse was 96, temperature 98° F., respiration 30. There was very marked general subcutaneous œdema. The skin and mucous membranes were rather pale, but the man was well-nourished and did not feel at all sick. A soft systolic murmur could be heard at the apex and base of the heart; the action of the heart was somewhat intermittent and irregular. The dropsy

increased for a few days and then gradually diminished. The record of the urine was as follows :

Date.	Quantity in Ounces.	Albumin.	Specific Gravity.	Urea in grains.
April 19.....	68	None	1024	608
" 20.....	42	Trace	1012	...
" 21.....	96	"	1022	340
" 22.....	150	"	1010	351
" 23.....	130	"	1026	280
" 24.....	84	"	1008	479
" 25.....	132	None	1014	672
" 26.....	136	"	1012	858
" 27.....	62	"	1014	410
" 28.....	44	Trace	1016	394
" 29.....	45	None	1014	...
" 30.....	...	"	1016	...

By May 1st the dropsy had entirely disappeared and the man was apparently well.

I have seen a number of hospital patients who unquestionably had kidney disease, but who had attacks of subcutaneous œdema after exposure, apparently not connected with their kidney disease, but caused by inflammation of the skin.

The ordinary treatment of dropsy is directed to the removal of the serum after it has transuded from the vessels. We try to get rid of the dropsy by sweating, by diuresis, or by purging. It is evident that a much more satisfactory treatment would be to prevent the transudation. If we could find remedies to destroy the irritating substances in the blood and tissues which cause the blood serum to transude, we would be able to prevent the dropsy instead of having to get rid of it.

#### ALBUMINURIA.

Since the time of Richard Bright the presence of albumin in the urine has been regarded as a proof of kidney disease both by physicians and by the laity. And in spite of all evidence to the contrary this is still the popular belief. It is true that any educated physician will now admit that albumin may be absent with kidney disease and present without it, but this admission is largely theoretical. In practice the old belief makes itself felt, and the presence of albumin is still looked for as the main evidence of disease of the kidneys.

The general belief concerning the albumin has been that it is removed from the blood by the kidneys just as urea or sugar is, and that if large quantities of it are removed from the blood the composition of this fluid is changed. A good deal of pains has been taken to discover why it is that diseased kidneys should excrete albumin.

As a matter of fact, the presence of serum albumin and serum globulin in the urine means that the blood serum, of which they are constituents, has become mixed with the urine. The simplest way in which this can happen is to have bleeding from the bladder or kidneys so that the blood and urine are mixed. The ordinary way is for the blood serum to transude from the kidney capillaries just as it does from capillaries in other parts of the body. Albumin in the urine, therefore, means the same thing as serum in a serous cavity, that there has been a transudation of serum from the vessels. To keep the matter clear in our minds, whenever we use the word albuminuria we should do so with the idea that it is a popular way of saying that the urine has blood serum mixed with it. In this way we will think of the kidney as we do of other parts of the body—as liable to exudations of serum either of inflammatory or of dropsical character. But, just as in the serous membranes the exudation is not in the membrane but in its cavity, so in the kidney the exudation is not into the parenchyma but into the tubules.

THE CAUSES of albuminuria, therefore, are the same as the causes of dropsies in all parts of the body :

1. *Changes produced by inflammation in the walls of the capillaries* which render them more permeable. In this way are produced the albuminuria of both forms of acute nephritis, of the severe forms of acute degeneration, of acute congestion, and of some of the cases of chronic nephritis with exudation.

2. *Changes in the composition of the blood*, causing either increased filtration or increased secretion of serum. This would account for the albuminuria of anæmia, of puerperal eclampsia without nephritis, of the mild cases of acute degeneration, of some of the cases of acute and chronic nephritis with exudation.

3. *Changes in the blood-pressure*. This would be the probable cause of the albuminuria in some of the puerperal cases, in chronic congestion, in some of the cases of chronic nephritis with exudation, and in chronic nephritis without exudation.

4. *Non-inflammatory changes in the walls of the capillaries*, ren-

dering them more permeable to the escape of serum. Such changes would account for the albuminuria which is found without inflammation of the kidneys, changes in the blood, or alterations in the blood-pressure.

THE SIGNIFICANCE of albumin in the urine depends, therefore, altogether upon its causation. As a symptom it may be compared to cough. It is well known that, while cough is a frequent symptom of disease of the lungs, yet its presence does not tell us what the disease of the lungs is, nor does it even tell us that there is necessarily disease of the lungs at all. Albuminuria, while it always means that the capillaries of the kidneys allow the blood serum to transude through their walls, does not tell us whether the causation of this transudation resides in the kidneys or outside of them. The study of this causation is practically a study of the causes of dropsy.

#### ALBUMINURIA WITHOUT DISEASE OF THE KIDNEY.

The examination of the urine by the physicians of life insurance companies, and by other physicians who have examined this excretion in cases of school children, of soldiers, and of other groups of persons, has brought out the fact that albumin is present in the urine in many persons who have no disease of the kidneys. These persons can be arranged in the following groups :

1. *Paroxysmal or Cyclic Albuminuria*.—The characteristic features of this form of albuminuria are : that the quantity of albumin is large, while casts are few or absent ; that if we examine the urine at regular intervals during the twenty-four hours there is a regular rise and fall in the quantity of albumin. The albumin begins to appear soon after rising in the morning, increases through the day, falls after going to bed, disappears at night, and reappears again the next morning. This regular cycle can be disturbed by changing the hours of rest, of meals, and of exercise. The rule is that the appearance of the albumin is favored by exercise and by eating, while rest in bed causes it to disappear. There seems to be no way of accounting for this form of albuminuria, except by supposing that there are changes in the composition of the blood, or in the walls of the renal capillaries.

The persons in whom this form of albuminuria is present are



regularly young males, who also suffer from more or less disturbance of the general health. The patients suffer from anæmia, lose flesh and strength, have headaches, neuralgic pains, bodily and mental languor, hysteria, and disturbances of the functions of the stomach, liver, and intestines. But there is a great difference in the patients as to how far these additional symptoms are developed. In some they are but trifling, in others they are well marked.

To distinguish these patients from those who have a true nephritis is by no means easy; the diagnosis may remain doubtful for months, and even then it is difficult not to make mistakes.

The treatment of these patients consists in the regulation of the diet and mode of life, and the management of the disturbances of digestion and of the condition of the blood. The diet should be liberal and varied, but all indigestible articles of food must be excluded. Massage, hot and cold baths, and regulated exercise are to be systematically carried out. A climate which admits of many hours' daily exposure to the open air and sunlight is to be preferred.

All disorders of digestion are to be remedied as far as possible.

The change in the composition of the blood is not marked; neither the quantity of hæmoglobin nor the number of blood-cells is much diminished; iron is of service in the treatment of the affection, but does not act as a specific.

The prognosis as regards the life and health of these patients is good, but it may be very hard to get rid of the albumin altogether.

2. *Dietetic Albuminuria*.—This occurs both in children and in adults. It may follow the ingestion of only certain kinds of food—cheese, pastry, and eggs; or of any kind of food; or of any food which is not properly digested; or it may occur when exercise follows immediately upon the ingestion of food. The quantity of albumin is small and there are few or no casts.

If this form of albuminuria is temporary, it is not a serious condition, but if the disposition to it persists, the patients are to be regarded with suspicion. They are also liable to temporary glycosuria; they may have well-marked functional disturbance of the liver; they may have the gouty disposition; or they may have cirrhosis of the liver, or chronic endarteritis.

The treatment consists in regulating the diet and exercise in the same way as in persons with the gouty disposition; in reliev-

ing constipation ; and in the use of the drugs which are likely to increase the production of bile.

3. *Albuminuria after Exertion*.—The exertion must be severe and prolonged, in long and fatiguing marches by soldiers ; prolonged contests in walking or running ; violent exercises such as boxing or wrestling. The quantity of albumin may be considerable and numerous casts may also be present. It seems probable that this form of albuminuria is due to a congestion of the kidneys caused by the exertion. After the cessation of the exertion the albumin regularly disappears within a few hours or days. But a repetition of such temporary congestions of the kidney might lead to the development of a true nephritis.

4. *Simple Persistent Albuminuria*.—These patients may for years have small quantities of albumin nearly every day, but not at all hours in the day. The albumin is not abundant, it often disappears after rest ; there may also be a few hyaline casts. The patients have no other symptoms of kidney disease, even when they are under observation for years. But one always feels anxious concerning such persons. Sooner or later they are apt to develop chronic nephritis, or endocarditis, or endarteritis.

#### URÆMIA.

It is well established that the principal function of the kidneys is to remove from the body a quantity of excrementitious substances. It is equally well established that a number of the diseases of the kidney interfere with this function and allow the excrementitious substances to accumulate in the blood and tissues. It is a matter of daily observation that persons who suffer from kidney disease exhibit symptoms of such a character as to give the idea that these persons are in some way poisoned. The sequence seems to be logical : disease of the kidneys, failure to eliminate excrementitious substances, accumulation of such substances in the blood and tissues, poisoning of the body by these substances, the development of symptoms due to the poisoning. To such a morbid process the name of "uræmia" can properly be given. So, in the year 1894, we find the following definition of uræmia in Dunglison's Medical Dictionary : "Certain morbid phenomena, implicating the nervous centres more especially, due to retention of excrementitious substances in the blood which are normally excreted by the kidneys, as in Bright's disease." And

this definition fairly represents the popular belief concerning uræmia.

Unfortunately it is not possible to dismiss the subject in this easy way. We are confronted with many contradictions difficult of explanation, and a review of the history of the subject shows that these difficulties have always been felt.

The simplest explanation of the phenomena of uræmia—that they are due to the presence of urea in the blood—has been contended for by many observers from the time of Christison down to the present moment. The proof has been derived from the examination of the blood in human beings, and from experiments on animals.

It has been demonstrated over and over again that the blood of persons suffering from uræmic attacks may contain a large excess of urea, that their serous effusions may contain large quantities of urea, and even that the urea may appear as a dry powder on the surface of the skin. It has also been shown that in a number of cases the outbreak of uræmic convulsions is preceded by a diminution in the excretion of urine and of urea.

The experiments on animals have consisted in injections of urea into the veins, in the introduction of urea into the stomach, and in abolishing the function of the kidneys by ligating the blood-vessels or the ureters.

The introduction of urea in considerable quantities into the veins or into the stomach is well borne by animals, provided that the kidneys perform their functions. The urea is eliminated with the urine. If, on the other hand, after the injection of urea into the blood the animal is entirely deprived of fluids, or the functions of the kidney are arrested by operation, then vomiting, diarrhœa, muscular contractions, and death regularly follow.

Ligature of the blood-vessels of the kidneys, or of their ureters, or extirpation of the kidneys is followed by an accumulation of urea in the blood and tissues. The animals have vomiting and diarrhœa, become stupid, and die.

The contradictions to this theory of uræmia were soon noted. Owen Rees was one of the first to call attention to the fact that prolonged anuria is not necessarily accompanied with renal symptoms. His illustrative case was a patient in whom one kidney was absent; the ureter of the other kidney became blocked by a calculus, and there was complete suppression of urine. The quantity of urea in the blood was much increased. The patient died,

but there were no uræmic symptoms. Cases of suppression of urine lasting for a number of days are not infrequent, and the ordinary experience has been that it is precisely in these cases that uræmic symptoms are absent, although death regularly follows.

More than this, Bartels and others have found that the blood drawn immediately after a uræmic attack may contain no excess of urea. It is a matter of ordinary experience that uræmic symptoms may come on in persons who are passing a normal quantity of urine of good specific gravity. So there seems to be no escape from the facts that complete suppression of urine is not regularly followed by uræmic symptoms, and that uræmic symptoms may occur without an excess of urea in the blood, or a diminution in the excretion of normal urine.

So far as the experiments on animals are concerned, they seem merely to show that urea in the blood does but little harm, and that abolition of the functions of the kidneys causes death.

It was to escape from some of these difficulties that Frerichs proposed the explanation that the cause of uræmic symptoms was poisoning by carbonate of ammonia. He taught that urea in excess in the blood did no special harm, but that if by the action of some ferment the urea was changed into carbonate of ammonia, then symptoms of intoxication would regularly follow. This theory, at one time popular, is now so entirely abandoned that it is not necessary to state the objections to it.

A modification of the theory of intoxication by urea is that of intoxication by urea and the other excrementitious substances of the urine together. The same affirmative and negative facts are to be found for this theory as for that of poisoning by urea alone. Normal uncontaminated urine injected into the veins of animals seems to do little harm unless the kidneys of these animals are injured by operation. If the kidneys are operated on, the animals die.

In human beings there are the patients with uræmic symptoms and an excess of excrementitious substances in their blood and tissues; the patients with uræmic symptoms, but without any excess of excrementitious substances; and the patients with anuria, an excess of excrementitious substances, and no uræmic symptoms.

A very important modification of the chemical aspect of the question is that made by Oppler and others. They hold that it

is an error to think that urea or any other constituent of the urine acts as a blood poison. Rather an interference with the functions of the kidneys must lead to a disturbance of the regular chemical changes in all parts of the body. Such an interference is followed by a change in the nutrition of the tissues which shows itself in loss of weight, in anæmia, and in disturbances of the functions of the brain. This way of looking at the subject is certainly a very rational one.

The opposition to all the chemical explanations of uræmia looks to changes in the blood-pressure as the exciting cause of uræmic attacks. The most complete theory of this kind is that of Traube.

This theory explains the occurrence of uræmic attacks as follows: The disease of the kidneys causes thinning of the blood serum, hypertrophy of the left ventricle of the heart, and an excess of blood-pressure in the arteries. If by any accidental circumstance the blood-tension is suddenly increased, or the blood serum still further thinned, œdema and anæmia of the brain are produced. The form of the uræmic attack will vary with the portion of the brain which is rendered anæmic or œdematous. If the cerebral hemispheres alone are involved the patient simply becomes comatose; if the central portions of the brain alone are affected there will be convulsions without coma; if both the hemispheres and the central portions of the brain are anæmic and œdematous, both convulsions and coma are developed.

Traube also states:

That he never saw an attack of uræmia in renal disease where the left ventricle of the heart was not hypertrophied, and where an increase of tension in the aortic system could not be demonstrated.

That the diluted state of the blood serum can be recognized by the pallor of the skin and mucous membranes and the presence of dropsical effusions.

That in every instance in which he examined the brain after death he could confirm the existence of anæmia and œdema.

That the presence of blood effusion within the cranial cavity in many of these cases confirms the suspicion that the abnormally high arterial blood-pressure to which these effusions owe their origin has also something to do with the production of the œdema which is present at the same time.

Experiments on animals have also shown that by ligating the

ureters, then the jugular vein on one side, and then injecting water into the carotid on the same side, general convulsions and coma can be produced. After death œdema of the brain without extravasation of blood is found.

The objections to Traube's theory are obvious: In patients who exhibit well-marked cerebral symptoms the specific gravity of the blood serum is not always lowered; the arterial tension is not always increased; neither anæmia nor œdema of the brain can always be demonstrated after death. These are facts which are soon ascertained by anyone who sees much of kidney disease.

The marked uræmic symptoms which occur at the close of pregnancy, and are known under the name of puerperal eclampsia, differ from ordinary uræmic attacks in that they may occur without marked structural changes in the kidneys. A variety of explanations have been offered as to their causation.

The older British and American obstetricians taught that puerperal convulsions were caused by determination of blood to the head—cerebral congestion. Traube's theory of altered blood, increased arterial tension with anæmia and œdema of the brain can be applied to the puerperal cases of uræmia as well as to those associated with kidney disease.

A retention in the blood of some toxic agent, with consequent poisoning of the blood centres, has been a favorite theory with many. The toxic material has been thought to be: urea, carbonate of ammonia, urea with kreatin and other excrementitious substances, or ptomaines produced by the growth of bacteria.

The convulsions are attributed by some to cerebro-spinal disturbance from peripheral stimulation quite independently of the kidneys. Others believe that the convulsions are due to blood poisoning, but that the renal disturbance which causes the blood poisoning is due to vasomotor spasm of the small renal vessels with consequent degenerative changes in the kidneys, the vasomotor spasm resulting from some reflex irritation.

It is also believed that some puerperal convulsions are simply acute epileptic attacks, the area of distribution of the sciatic nerve being the epileptogenic zone.

More recently attention has been called to the probability that the so-called uræmic symptoms are due to a poison in the blood, but that this poison is not due to any disturbance of the

function of the kidneys. This idea is only a theory, but it offers a promising field for study. It may very well be that we must look for the cause of these symptoms altogether outside of the kidneys.

It is evident from what has been said that there is no entirely satisfactory way of accounting for the so-called uræmic symptoms. At the present time the only very useful thing to do is to try and state as clearly as possible the conditions of the problem which we wish to solve.

THE SYMPTOMS which it is customary to call uræmic are :

1. *Headache and sleeplessness*, which come on in attacks of short duration, or may be continued for many weeks. The headache may be of mild type or very severe. In extreme cases the pain is so severe and the sleeplessness so distressing that the patients are almost maniacal. These symptoms may accompany :

(a) Puerperal Eclampsia either with or without Nephritis. At the time of the attack the urine is diminished in quantity but of good specific gravity. The arteries are full and tense, the heart's action is exaggerated, the veins are congested. The headache can be relieved by the birth of the child, by general blood-letting, by morphine, and by the drugs which dilate the arteries.

(b) Acute Exudative and Acute Productive Nephritis. At the time of the attack the urine is often, but not always, diminished; its specific gravity is good. The arteries are full and tense, the heart's action is exaggerated. The headache can be relieved by the arterial dilators, by morphine, by purging, by sweating. General blood-letting can but seldom be used.

(c) Chronic Nephritis with Exudation. The urine is often diminished, but may be increased in quantity, its specific gravity is low. The condition of the arteries and heart is not constant. The arteries may be full and tense, or small and tense, or full and soft, or small and feeble. The heart's action may be exaggerated or feeble; the valves may be diseased, or the left ventricle hypertrophied. The headache can sometimes be relieved, but late in the disease nothing will control it. The arterial dilators are of use only when the pulse is tense. The cardiac stimulants may do good when the heart's action is feeble. Thorough daily sweating is sometimes efficient. Purging can give temporary relief. Opium may be the only drug that is of

any use. Mild cases can be relieved by improving the action of the digestive tract.

(d) *Chronic Nephritis without Exudation.* The headache and sleeplessness are especially frequent and severe with this form of nephritis. The urine is often diminished, but may be increased, or normal in quantity; its specific gravity is low. The condition of the heart and arteries is liable to the same variations as in chronic nephritis with exudation, but a full, tense pulse and an hypertrophied left ventricle are more regularly present. The first attack of headache can usually be relieved, but each successive attack is more difficult to manage.

2. *Hemiplegia and Aphasia.*—These two symptoms, which may occur separately or together, are seen in patients who have chronic nephritis without exudation, and in women with puerperal eclampsia. The invasion of the hemiplegia is sudden and it is usually accompanied by coma. There is loss of motion alone, or of both motion and sensation. The hemiplegia, coma, and aphasia may continue up to the time of the patient's death, or disappear after a few days. In the latter case the patient may have several such attacks. In chronic nephritis the hemiplegia may occur either early or late in the course of the disease. In these patients chronic endarteritis, especially of the cerebral arteries, is very regularly present. I think that it is probable that the hemiplegia is due to the endarteritis, rather than to the kidney disease.

The treatment of this condition is not satisfactory. If there is well-marked arterial tension it may be proper to try and reduce it, otherwise it is better not to interfere.

3. *Sudden Blindness.*—Besides the loss of vision due to nephritic retinitis, there may be a sudden blindness which lasts for hours or days. In these patients no anatomical changes in the eyes have been discovered. This form of blindness is not uncommon in puerperal eclampsia; it occurs in a moderate number of the cases of chronic nephritis. We do not understand the nature or treatment of this blindness; fortunately it only lasts for a short time.

4. *General Epileptiform Convulsions.*—These have always attracted much attention as one of the most terrible and dangerous of the results of kidney disease. They may accompany:

(a) *Puerperal Eclampsia*, either with or without nephritis, coming on either before, during, or after labor. At the time of



the attack the urine is sometimes diminished or suppressed, sometimes of normal quantity. The arteries are regularly full and tense, the heart's action is exaggerated, the skin is congested. It seems to be generally conceded that in these patients the convulsions are not all due to the same cause. There is also a substantial agreement as to the best methods of treatment. General blood-letting for the patients with excessive venous congestion, chloroform inhalations for the irritable patients, and the drugs which dilate the arteries, are the routine treatment.

(b) Both Forms of Acute Nephritis. In children suffering from acute nephritis convulsions are of quite frequent occurrence, even when the disease is not of severe type. So many children recover after one or more convulsions that they are not grave symptoms. In adults convulsions do not occur nearly as often as in children, but when they do occur the patients are much more likely to die. In children general blood-letting can very seldom be practised; in adults there are a few cases in which it is appropriate. In most of the patients the drugs which dilate the arteries give altogether the best results.

(c) Chronic Nephritis with Exudation. The convulsions belong to the advanced cases of the disease, to the patients who are dropsical, anæmic, and apparently thoroughly poisoned with excrementitious substances. The heart's action and pulse are feeble. In these patients it is difficult, or impossible, to control the convulsions. The most efficient plan seems to be the daily sweating with the hot pack.

There are cases of chronic nephritis with exudation in which an exacerbation of the inflammation takes place, and the patients then behave much as if they had an acute nephritis.

(d) Chronic Nephritis without Exudation. Convulsions are of frequent occurrence early in the disease as well as late; indeed, in many persons the attack of convulsions is the first symptom of the nephritis. Many of the patients have hypertrophy of the left ventricle of the heart and chronic endarteritis in addition to the nephritis. Common as these attacks are, their causation is most obscure, for there can be no question that the liability to the convulsions is not at all in proportion to the failure of the functions of the kidneys. Certainly a marked increase in arterial tension is the rule with these attacks, and the control of the convulsions is in proportion to the success in dilating the

arteries. But there are cases in which we are not able either to dilate the arteries or to control the convulsions; and there are cases in which the convulsions continue although the pulse becomes rapid and feeble. It is for the convulsions with this form of nephritis that hypodermics of morphine are of so much efficacy for a time. It must be admitted that when a patient with chronic nephritis begins to have convulsions death is not far off.

5. *Contractions of Groups of Muscles.*—These are of common occurrence in the severe forms of acute and chronic nephritis and in puerperal eclampsia. Very often they merely precede an attack of general convulsions. They are best marked and of longest continuance in the advanced cases of chronic nephritis with exudation.

6. *Delirium and Coma.*—They come on suddenly in attacks, either associated with convulsions or by themselves; or they are developed slowly and gradually and continue for a considerable length of time. The attacks belong to the severe cases of acute nephritis, to the exacerbations of chronic nephritis with exudation, and to the ordinary cases of chronic nephritis without exudation. The gradual development of delirium and coma is apt to continue, although sometimes with intervals of improvement, up to the time of the patient's death. They are very often seen in the advanced stages of both forms of chronic nephritis.

7. *Vomiting* is seen in many cases of nephritis. It is evidently due to a number of different causes. In acute nephritis the vomiting seems to be of the same character as that which may occur with an acute inflammation of any part of the body. In chronic nephritis the vomiting may be due to chronic gastritis; or the stomach, like the intestine, gets rid of some of the accumulated urea and serum.

Besides these forms of vomiting there is a special and aggravated form which belongs regularly to the cases of chronic nephritis without exudation. It is usually accompanied by a marked increase of arterial tension. The vomiting is frequent, distressing, and may continue for days. The most efficient means of controlling it are chloral hydrate by the rectum in twenty-grain doses, or hypodermic injections of morphine.

8. *A Rise of Temperature.*—In acute nephritis, as in any other acute inflammation, there may be a febrile movement. As a rule the temperature is not high, and falls to the normal within a week. But in children temperatures of 104° or 105° F. may be reached.

In chronic nephritis the severe attacks of cerebral symptoms—headache, convulsions, delirium, coma, hemiplegia—are not infrequently accompanied with a considerable rise of temperature. I have seen it as high as  $109^{\circ}$  F., and yet the autopsies show no reason for the febrile movement.

9. *Dyspnœa* is one of the most frequent and interesting of the symptoms of nephritis. There is a dyspnœa due to fluid in the pleural cavities, or to œdema of the lungs; a dyspnœa due to the pressure on the diaphragm by fluid in the abdomen; and a dyspnœa due to bronchitis or to contraction of the bronchi; but the most important form of dyspnœa is one which is independent of all these causes, and is directly caused by disturbances of the circulation. It belongs to both forms of chronic nephritis, but is more common with nephritis without exudation. The patients, as a rule, in addition to the nephritis have pulmonary emphysema, chronic endarteritis, hypertrophy of the left ventricle, or chronic endocarditis. Not infrequently one or more of these lesions is much more advanced and apparently more important than the kidney disease. Especially is this the case with chronic endarteritis, which gives many of the most marked examples of this form of dyspnœa with but very little nephritis.

The dyspnœa may come on at any time in the course of a nephritis; very often it is the first symptom which causes the patient to seek medical advice. It begins with attacks which at first are brought on by bodily and mental exertion, or come on of themselves at an early hour in the morning. In none of the attacks do we hear the characteristic breathing of bronchial asthma. The attacks at first only last for an hour or so, and during the rest of the day the breathing is comfortable. But even in these mild attacks the patients cannot lie down. As time goes on the attacks become more frequent and of longer duration. Finally comes the terrible period when the breathing is always bad, the patients cannot lie down at all, and yet go on living for weeks and months.

The treatment of this dyspnœa is often for a time extremely satisfactory. The patients are enabled to live and work in comfort for many years. But each succeeding attack is harder to control than the first, and finally there comes a time when everything fails and the dyspnœa continues although the patient is stupefied with drugs.

The object of treatment is to relieve the disturbances of the

circulation ; if this can be done the dyspnœa is also relieved. To effect this the most exact study of the circulation is required. For each patient the character of the heart's action and of the pulse, both relatively and separately, must be determined. Based on this knowledge is the intelligent use of cardiac stimulants and sedatives, of arterial dilators, of regulation of the functions of the stomach, liver, and intestines, of rest or exercise. There is no one plan of treatment for all, nor even one plan for the same patient in all attacks.

*Increased Arterial Tension.*—This is not always classed with the uræmic symptoms. It is, however, one of the most frequent and important of the symptoms of chronic nephritis ; and it is by it that many of the so-called uræmic symptoms are produced.

It has been believed that this increased tension of the blood in the arteries is due to chronic changes in the walls of the arteries and capillaries which interfere with the passage of the blood through them. The explanation is very probably true up to a certain point, but it does not account for the attacks of increased arterial tension which come and go within a few hours. I do not see how these can be produced except by the temporary contraction of arteries which have a well-developed muscular coat, such as the radial artery. I think that it is possible to demonstrate after death in such arteries an hypertrophy of the muscular coat, in patients who have had many attacks of increased arterial tension.

If this is admitted, then we have to find a reason for the attacks of contraction of the arteries which last for hours or for weeks, and which can often be controlled by the drugs which dilate the arteries.

Such attacks of contraction of the arteries occur with :

Attacks of angina pectoris.

Chronic endocarditis.

Chronic arteritis.

Pulmonary emphysema.

Chronic nephritis.

It seems as if such a contraction of the arteries must be due to some irritating substance in the blood. But whether there is only one poison which acts in this way or several poisons, and how such poison or poisons are produced, we do not know.

## ACUTE CONGESTION OF THE KIDNEYS.

## DEFINITION.

A temporary congestion of the blood-vessels of the kidney, which may be accompanied with exudation of serum and escape of red blood-cells.

## ETIOLOGY.

Acute congestion is caused by the ingestion of certain poisons, by extirpation of one of the kidneys, by severe injuries inflicted on any part of the body, by surgical operations, especially those on the bladder and urethra, and by over-exertion.

## MORBID ANATOMY.

It is but seldom that we are able to obtain human kidneys in the state of acute congestion, for the condition is not usually a fatal one. In animals, however, the condition can be produced experimentally by cantharidin. It is found that the kidneys are enlarged, that the veins, capillaries, and Malpighian tufts contain an increased quantity of blood, and that the epithelial cells of the cortex tubes are flattened.

## SYMPTOMS.

Acute congestion may occur in kidneys previously normal, or in those already diseased.

The urine is diminished in quantity or suppressed ; its specific gravity is unchanged ; it contains blood, albumin, and casts.

(1) *Congestion Caused by the Ingestion of Poisons.*

Cantharides given internally, or used in blisters or ointment, is rather a frequent cause of acute congestion of both the kidneys and the bladder.

The urine is diminished in quantity ; it is passed frequently, in small quantities, with much pain ; or it is retained. It contains albumin, a few casts, and blood. Sometimes large, jelly-like coagula are formed in the bladder.

The patients may have a moderate rise of temperature, pain in the back, abdominal pain, nausea, and vomiting, diarrhœa, more or less prostration, delirium, and stupor. The severity of the symptoms depends on the quantity of the drug absorbed. In the bad cases of poisoning the condition of the kidneys is not that of hyperæmia, but of actual inflammation.

When there is only hyperæmia the patients are sick for a few days, and the urine soon returns to its natural condition. Turpentine produces symptoms like those of cantharides.

*Treatment.*—If the poison has been taken into the stomach, that organ is to be emptied and washed out. Warm baths, or a hot pack, and the use of small doses of opium are of service. Camphor in doses of from 2 to 5 grains every three hours has been recommended in cantharides poisoning.

### (2) *Congestion following the Removal of One Kidney.*

The urine is scanty or suppressed, and contains albumin and casts. The patients are in a condition of prostration which is very alarming. They may remain in this condition for a few days and then recover, the urine returning after a time to its natural condition; or they become more and more feeble, pass into the typhoid condition with mild delirium, and die.

*Treatment.*—The patients are to be kept perfectly quiet in bed, on a fluid diet. They should have one or two long hot packs every day, so as to produce congestion of the skin and profuse perspiration.

### (3) *Congestion of the Kidneys after Injuries or Surgical Operations.*

It is well known that any operation on the bladder or urethra, even the passage of a catheter, may be followed by suppression of urine, great prostration, and death within forty-eight hours, and that after death no lesion is found except congestion of the kidneys. These cases are not to be confounded with the cases of septic infection and fever, which may also follow operations on the urethra and bladder.

It is not so well known that surgical operations on any part of the body are occasionally followed by suppression of urine, congestion of the kidneys, and death. We do not know that death in all these cases is caused by the congestion of the kid-

neys, but there can be no question that congestion of the kidneys is produced in this way.

*Treatment.*—No satisfactory treatment has yet been found for these patients; in fact, the very short time which intervenes between the operation and death hardly gives time for treatment.

#### (4) *Acute Congestion after Over-exertion.*

Prolonged marches, violent gymnastic exercises, contests of walking and running prolonged over several days, may be followed by the excretion of such a quantity of albumin and casts in the urine as to indicate a temporary congestion. I do not know of any post-mortem observations which corroborate this belief. There are no other symptoms besides the changes in the urine, and these disappear after a few days' rest.

### CHRONIC CONGESTION OF THE KIDNEYS.

There are a number of morbid conditions which interfere with the circulation of the blood in the aortic system in such a way that the blood accumulates in the veins and is diminished in the arteries. The most common of these conditions are: chronic inflammation of the aortic and mitral valves, dilatation of the heart, aneurism of the arch of the aorta, pulmonary emphysema, and large accumulations of fluid in the pleural cavities.

In pulmonary emphysema the disturbances of circulation are confined to the cases in which there is obstruction to the passage of blood through the lungs, dilatation and hypertrophy of the right ventricle, and then venous congestion of the aortic system. More or less dropsy is regularly developed at about the same time as the congestion of the kidneys.

Large accumulations of fluid in the pleural cavities, if they remain for any length of time, may produce well-marked chronic congestion. This is denied by Bartels, who says that he has never known congestion or any serious disturbance of the renal functions to occur as a result of pleuritic exudations. I have, however, seen congestion produced in this way a number of times. I think that it is a lesion of consequence to the patient, and I believe that it furnishes an additional reason for the early removal of fluid from the pleural cavities.

## ETIOLOGY.

By far the most common cause of chronic congestion of the kidneys is disease of the heart. So long as a heart with chronic endocarditis, or myocarditis, or dilatation, is able, in spite of its damaged state, to carry on the circulation fairly well, no secondary changes in the kidneys are produced. But as soon as the blood accumulates in the veins to any considerable extent the kidneys suffer. One of three things regularly happens to them : either chronic congestion, or chronic degeneration, or chronic nephritis is developed. It is also necessary to remember that chronic endocarditis and chronic nephritis often exist in the same person, although neither one of them is secondary to the other.

## MORBID ANATOMY.

The kidneys are of medium size, or rather large. Their weight is increased, somewhat out of proportion to the increase in size. The color is dark-red, the consistence is very hard, the surfaces are smooth, the capsules are not adherent. The congestion is most marked in the veins of the pyramids; they contain an increased quantity of blood, and are often dilated. The capillaries of the cortex are also congested, but it is rather exceptional to find them dilated. The epithelium of the convoluted tubes is swollen, and the separate cells of which it is composed are more evident. Or, instead of this, the epithelium is much flattened, so that the lumen of the tube is larger. I think that this flattening, of the epithelium belongs to the kidneys which give urine containing a good deal of albumin.

The most constant and characteristic change is in the glomeruli. The capillaries which make up the glomerulus are dilated, with more or less thickening of their walls. So far as I know this change in the glomeruli is constant and persists, even if the congestion is succeeded by a true nephritis.

While the congestion often persists up to the time of the patient's death, it may, instead of this, be followed by an acute or a chronic nephritis.

If there is an acute nephritis albumin is present in considerable quantities in the urine. After death the glomeruli, in addition to the dilatation of their capillaries, show an increase in the



size and number of the cells which cover them. The epithelium of the convoluted tubes is flattened.

If there is a chronic nephritis the specific gravity of the urine falls and the excretion of urea is diminished. The nephritis follows the anatomical type of a chronic nephritis without exudation, but the dilatation of the capillaries of the glomeruli persist.

#### SYMPTOMS.

Of the persons who die with chronic congestion of the kidneys a large number present marked symptoms during life, but it is difficult to determine how largely these symptoms are due to the congestion of the kidneys.

A congestion of a few weeks' duration, such as we see with large effusions of fluid in the pleural cavities, seems to give no renal symptoms and no changes in the urine.

The question is complicated by the fact that, with chronic endocarditis and pulmonary emphysema, any one of the following kidney lesions may coexist :

1. Chronic congestion.
2. Chronic congestion followed by chronic exudative nephritis.
3. Chronic congestion followed by chronic nephritis without exudation.
4. Chronic degeneration.
5. Chronic degeneration followed by chronic nephritis with exudation.
6. Chronic nephritis with exudation not preceded by congestion.
7. Chronic nephritis without exudation not preceded by congestion.

Here are seven different kidney lesions, each one of them frequently associated with cardiac disease.

In bad cases of heart disease the ordinary symptoms are : dyspnœa; cough and expectoration, general dropsy, vomiting, headache, delirium, convulsions, coma, anæmia, loss of flesh and strength.

It is always difficult, and often impossible, in any given case of heart disease to determine certainly which kidney lesion exists. The general rules which we follow are these :

*Chronic Congestion.*—The cardiac symptoms not as severe ;

dropsy often present ; the quantity of the urine moderately diminished, its specific gravity normal or higher than normal, albumin in very small quantities, few or no hyaline casts.

*Chronic Congestion followed by Chronic Nephritis with Exudation.*—The cardiac symptoms more severe ; the dropsy well marked ; the urine scanty, its specific gravity normal or lowered, a large quantity of albumin, and casts in variable number.

*Chronic Congestion followed by Chronic Nephritis without Exudation.*—The patients are liable to have attacks of contraction of the arteries. The dropsy is not as constant. The quantity of the urine varies, sometimes above, sometimes below the normal. Its specific gravity is low. There is but little albumin except when the arteries are contracted. Loss of flesh and strength are marked features.

*Chronic Degeneration.*—The cardiac symptoms are severe ; dropsy is regularly present, but not always marked. The quantity of the urine varies, its specific gravity is unchanged, there is but little albumin. The patients are feeble and anæmic with a prolonged period of scanty urine, delirium, stupor, and the typhoid state.

*Chronic Degeneration followed by Chronic Nephritis with Exudation.*—The cardiac symptoms are severe and the dropsy well marked. The quantity of urine is small, its specific gravity is rather high than low. Albumin is present in large quantities, casts are not as constant. The patients are feeble, anæmic, and get worse rapidly.

*Chronic Nephritis either with or without Exudation, not preceded by Congestion.*—In these patients there is the ordinary history of chronic nephritis lasting for months or years. Then, as the endocarditis advances, the heart's action becomes worse and the circulation is disturbed, the different cardiac symptoms are added.

*Treatment.*—Although we are unable to remove the mechanical interference with the circulation, yet the condition of the patients can often be very much improved. The main indications are to improve the character of the circulation and to remove the dropsy. In order to improve the circulation it is absolutely necessary to study the character of the heart's action, to appreciate the organic changes in the heart, and to determine whether the walls of the arteries are thickened, and whether there is an increase or diminution of the arterial tension. It is unfortunate

that there is no instrument to tell us certainly what the arterial tension is; we have to depend upon the sense of touch. This, however, can be educated, and a physician is not competent to manage cardiac cases if he cannot appreciate changes in arterial tension.

In many patients with an irregular heart, dyspnoea, and dropsy, simple rest is of the greatest service. This is especially marked in hospital patients who have tried to work up to the time of their admission. It is often a good rule to give no medicine to such patients until we see how much they improve with rest alone.

The regulation of the food is of importance. It is to be remembered that, while gastric indigestion and flatulence make the heart-action worse, on the other hand meat is one of the best cardiac stimulants. Most patients are better for meat in some form at least once a day.

Tumultuous and exaggerated heart-action may be due to contraction of the arteries and require such drugs as nitro-glycerin or chloral hydrate. It may be the expression of a heart which is really feeble and requires digitalis. It may mean a real exaggeration of the muscular action of the heart and be benefited by aconite, belladonna, and the local action of cold. A feeble heart without contraction of the arteries calls for cardiac stimulants; digitalis, strophanthus, and caffeine are the best. But it is to be remembered that with aortic and mitral stenosis, myocarditis, and diseased coronary arteries, there comes a time when cardiac stimulants make the heart's action worse instead of better. This is the time for the use of opium. Small doses of codeine or of morphine give the patient more relief than can be obtained in any other way.

In many patients the condition of the heart and arteries varies from day to day, so that there have to be many corresponding changes and combinations of the drugs—the cardiac stimulants, the arterial dilators, and the opium.

The dropsy is more or less favorably affected by the rest in bed and the regulation of the circulation. It can also be reduced by the hot pack and the hot-air bath.

For very extensive cardiac dropsies, with only chronic congestion of the kidney or but little nephritis, calomel is the most efficient diuretic. Three grains of calomel with  $\frac{1}{16}$  of a grain of morphine, four times a day for four days, is the dose. The

mouth and gums are to be kept very clean. If the drug acts properly there is but little effect on the bowels and no salivation, but on the third day the quantity of urine increases and remains large for several days. The dropsy diminishes and may disappear altogether.

#### ACUTE DEGENERATION OF THE KIDNEYS.

##### DEFINITION.

An acute change in the kidneys, characterized by degeneration or death of the epithelial cells of the tubules, to which may be added an exudation from the blood-vessels.

*Synonyms.*—Acute Bright's disease ; Parenchymatous Nephritis ; Parenchymatous Degeneration.

##### ETIOLOGY.

The introduction of certain poisons into the body is regularly followed by changes in the cells of the viscera. The poisons which exert this effect are ordinarily mineral poisons, such as arsenic, mercury, and phosphorus ; or the poisons of infectious diseases, such as diphtheria, typhoid fever, etc. According to the quantity and virulence of the poison received into the body, there are more or less marked changes produced in the cells of the viscera.

Small doses of such poisons, acting only for a moderate length of time, produce simple swelling of the cells. The cells are swollen, more opaque, more coarsely granular. They are not dead, nor broken down, nor do they contain any new substances ; the change in their appearance is due to the swelling of the network which forms a part of every cell. Under these circumstances there are either no changes at all in the blood-vessels of the viscera, or a slight congestion, with, perhaps, a little exudation of serum.

Larger doses of such poisons, or more virulent poisons, or a longer duration of the action of a poison, are attended by the deposition in the cell-bodies of granules of albuminous matter and globules of fat. At the same time there is a change in the nutrition of the cells, and they are often broken and disintegrated. Under these conditions there may be considerable congestion of the vessels and an exudation of serum.

Very large doses of such poisons cause the death of the cells of the viscera, a death which may take the form of coagulation-necrosis or of disintegration and breaking down of the cell. With these changes there will often be an excessive congestion of the vessels and a large exudation of serum.

As the kidneys are excreting organs it is rather natural to think that the substances which cause degeneration of the renal epithelium do so because they are excreted by the kidneys. But, as the same poisons produce similar degeneration in many other parts of the body, it seems more probable that the effect of the poison is produced in the same way that it is in the nerves, the muscles, the liver, and the spleen.

The well-known fact that temporary cutting off of the arterial blood from the kidneys in animals is followed by degeneration or death of the renal epithelium, has led to the idea that degeneration of the kidneys, especially in cholera, is due to ischæmia. This seems possible, but it is a theory not at all applicable to most cases of acute degeneration.

It is curious that so many different poisons should act in the same way. There seems to be nothing in common between the poison of corrosive sublimate and that of yellow fever, and yet the changes in the kidneys are practically the same.

The inorganic poisons, arsenic, etc., act according to the size of the dose taken. A small dose produces only moderate degeneration of the renal epithelium, a large dose causes extensive necrosis with considerable exudation of blood serum.

The toxins of the different infectious diseases vary as to the activity of their effect on the renal epithelium, as to the time in the disease when the degeneration takes place, and as to the frequency with which true nephritis is produced instead of acute degeneration. It is a question of much importance whether the same toxin produces degeneration or nephritis according to its dose, or whether two or more different toxins are necessary. In scarlatina and diphtheria, for example, the rule is that acute degeneration comes in the early days of the disease, acute exudative nephritis in the late days of the disease, and acute productive nephritis just after the close of the disease. Does this mean three different toxins, or that the same toxin varies at different stages of the disease, or that the only difference is in the dose?

For clinical purposes the recognition of the fact that acute

degeneration is the ordinary lesion of the infectious diseases, is of much practical importance.

#### MORBID ANATOMY.

The gross appearance of the kidney varies with the extent of the degeneration. In the ordinary mild cases, such as accompany pneumonia, the kidney is a little larger, the cortical portion a little thicker and paler. In the severe cases, such as accompany acute yellow atrophy of the liver, the kidney is considerably enlarged and more or less congested.

The changes in the renal epithelial cells are : a simple swelling of the cell-body, causing it to look larger and more opaque and to take on irregular shapes ; an infiltration of the cell-bodies with granules of albuminoid matter and fat ; a death of the cells, which may take the form of coagulation necrosis or of a disintegration of the cell-bodies ; a desquamation of the dead cells so that the tubes are filled with them ; a formation of hyaline masses in the cells ; a growth of new cells to take the place of the dead epithelium. All these changes are most marked in the convoluted tubes.

In the kidneys with extensive necrosis of the epithelium there may also be congestion of the blood-vessels and casts in the tubes.

#### SYMPTOMS.

With the severe acute degeneration which follows the ingestion of large doses of arsenic, mercury, or one of the other inorganic poisons, the urine is diminished in quantity, or suppressed ; it contains albumin, casts, and blood ; its specific gravity is unchanged. At first the general symptoms belonging to the poison predominate, but as these subside the patients continue to live and suffer more or less severely from the degeneration of the kidney. They become feeble, pass into the typhoid state, and often die.

The acute degeneration which accompanies the infectious diseases such as scarlatina, diphtheria, typhoid fever, pneumonia, etc., is for the most part of mild type and gives no symptoms except the presence of a little albumin and a few casts in the urine. It is of importance to recognize the frequency with which this affection of the kidney occurs, the slight injury which it inflicts on the patient, and the completeness with which the kidney

lesion disappears after the recovery from the primary disease. Much unnecessary anxiety is often felt by physicians because in a case of pneumonia or some other infectious disease they find albumin and casts in the urine. There are fears not only of death from the primary disease, but of the subsequent development of "Bright's disease." If the albumin and casts are due to degeneration of the kidneys anxiety is needless. The course of the primary disease will not be changed. If the patients recover their kidneys return to their normal condition.

With yellow fever, with acute yellow atrophy of the liver, and occasionally with the other infectious diseases the degeneration is of intense type, with the death of a large part of the renal epithelium and exudation from the vessels. In such cases the urine is scanty or suppressed. It contains albumin, casts, and blood. The patients are very ill, they may have convulsions, delirium, or stupor; they often die. But it is hard to tell how many of their symptoms are due to the complicating kidney lesion.

#### TREATMENT.

So far as the degeneration of the epithelium is concerned, we know of no treatment which is likely to affect it favorably. But in the severe cases with congestion of the kidneys and scanty urine, it seems to be good practice to use the hot-air baths or the hot pack.

It is evident that acute degeneration of the kidney can be sharply distinguished from all other forms of kidney disease. It is always produced by the introduction of some poison into the body. It is not accompanied by dropsy, contraction of the arteries, or by other renal symptoms. It is not usually dangerous to life. It is a temporary condition not followed by any chronic kidney disease. If the patient recovers from his poisoning the kidneys return to their normal condition.

#### CHRONIC DEGENERATION OF THE KIDNEY.

##### DEFINITION.

A chronic disease of the kidneys characterized by degenerative changes in the renal epithelium.

*Synonyms.*—Chronic Bright's disease, Chronic Parenchymatous Nephritis, Fatty Kidney.

## ETIOLOGY.

The same mechanical obstructions to the circulation—heart disease, pleuritic effusions, etc.—which produce chronic congestion, can, instead of this, produce chronic degeneration of the kidney.

It is said that anæmia of the kidneys produces degeneration of the renal epithelium. Experiments upon animals show that this view is theoretically possible. It may be that the degeneration of the kidneys seen in old and feeble persons is due to a diminished blood-supply, but we can hardly speak with certainty on this point.

Chronic diseases, such as phthisis and cancer, are followed by chronic degeneration of the kidneys.

There is a group of cases in which, although the health of the patients is not good, it is not easy to fix on a definite cause for the chronic degeneration.

Apparently many of the authors who describe a "chronic parenchymatous nephritis" include under this head both chronic degeneration and chronic nephritis.

The matter is further complicated by the fact that kidneys may be in the condition of chronic degeneration for some time, and then become further altered by a chronic nephritis with exudation, and by waxy degeneration of the glomeruli.

## MORBID ANATOMY.

If the degeneration follows heart disease the kidneys are large, weighing together from sixteen to twenty ounces. Their surfaces are smooth; the cortical portion is thickened, of pink or white color, the pyramids are red. The gross appearance is that of the so-called large white kidney. The epithelium of the cortex tubes is swollen and coarsely granular. The capillaries of the glomeruli are dilated, with more or less thickening of their walls. The veins in the pyramids are congested. There are no changes in the stroma, or in the arteries.

If the degeneration follows phthisis, cancer, or any wasting disease, the kidneys are usually large, with a white or yellowish cortex. There are no changes except in the cortex tubes. In these the epithelial cells are either coarsely granular or infiltrated with fat.



If the degeneration occurs in old people, or without discoverable cause, the kidneys may be either large and white, or of the size and appearance of a normal kidney, or small and red. There are the same degenerative changes in the epithelium of the cortex tubes, with no lesions in the stroma or the glomeruli.

#### SYMPTOMS.

With the degeneration caused by heart disease the quantity of the urine varies with the changes in the action of the heart and the contraction of the arteries, sometimes abundant, sometimes scanty, sometimes suppressed. The specific gravity is not diminished, nor is the proportion of urea to the ounce diminished. Albumin and casts in small quantities are more frequently present than with chronic congestion. While it is difficult to separate the kidney symptoms from the heart symptoms, yet one has the impression that this kidney lesion is more serious than chronic congestion, and has its effect in increasing the loss of nutrition and the anæmia.

If the degeneration is followed by chronic nephritis with exudation, albumin appears in the urine in large quantities; the anæmia and dropsy are very marked, and the patients get worse rapidly.

When the degeneration accompanies chronic diseases, such as phthisis or cancer, the urine remains normal, or from time to time contains a little albumin and a few casts. The patients are so ill with their primary disease that the secondary degeneration of the kidneys is hardly appreciable during life.

The group of cases in which the degeneration occurs without discoverable cause is interesting, for the kidney lesion may be the cause of death. It is a difficult group to study, because the cases are not very numerous, and the clinical histories and autopsies are apt to be dissociated. The autopsies are for the most part in hospital patients with short and imperfect histories; while the clinical histories are of private patients on whom it is difficult to obtain autopsies. I think, however, that it can be said the urine remains normal, or from time to time contains a little albumin and few casts. Neither dropsy, nor a pulse of high tension, nor a hypertrophied left ventricle, nor acute uræmic symptoms are present.

The patients gradually, month after month, lose flesh and

strength and become more or less anæmic. They may have a variety of digestive disturbances. The course of the disease is slow, sometimes interrupted by periods of improvement, but regularly getting worse from year to year. Finally the patients are so feeble that they remain in bed; they develop alternating delirium and stupor, and so die.

The diagnosis in these cases is extremely difficult. They resemble cases of malignant disease in which no tumor can be found, and cases of chronic nephritis with normal urine and no changes in the heart or arteries.

#### TREATMENT.

It is not easy to find means to influence chronic degeneration of the renal epithelium. The best that we can do for the patients is to stop all vicious habits, to regulate the diet and mode of life, and to relieve the disturbances of circulation.

### ACUTE EXUDATIVE NEPHRITIS.

#### DEFINITION.

An acute inflammation of the kidneys, characterized by congestion, exudations of the blood-plasma, emigration of white blood-cells, diapedesis of red blood-cells, to which may be added changes in the renal epithelium and in the glomeruli.

*Synonyms.*—Acute Bright's Disease; Parenchymatous Nephritis; Tubal Nephritis; Desquamative Nephritis; Catarrhal Nephritis; Croupous Nephritis; Glomerulo-nephritis.

#### ETIOLOGY.

Acute exudative nephritis is frequently a primary inflammation, occurring either after exposure to cold or without discoverable cause. It may complicate any one of the infectious inflammations or diseases, but is especially common with scarlet fever. It is one of the forms of nephritis which are caused by pregnancy.

Acute exudative inflammation in any part of the body seems to be caused by local irritation, by the presence of irritating substances in the blood, and by changes in the circulation of the blood and in the inflamed portion of the body. Pathogenic bac-

teria are sometimes present, sometimes absent in the inflamed tissue.

Unquestionably, all the infectious diseases are often complicated with inflammations of different parts of the body. The probable causes of these are the chemical poisons produced by the growth of the pathogenic bacteria belonging to each disease. It seems also that the poison of each disease has a preference for particular portions of the body, In rheumatism the joints and heart are regularly inflamed; in measles, the bronchi; in scarlet fever and diphtheria the throat and the kidneys.

As regards the presence of bacteria in the kidneys themselves as exciting causes of inflammation, our knowledge is uncertain.

Whether nephritis in puerperal women and after exposure to cold is due to disturbances of circulation or to some poison in the blood is not certain.

There are marked differences in the severity of different cases of nephritis. The exudation of serum is larger in one case, the emigration of white blood-cells in another. In one kidney the epithelial cells are contracted, in another they are swollen and degenerated. The glomeruli are much more changed in some kidneys than in others. How closely these differences in the lesions correspond to differences in the causation of the nephritis, we do not know.

#### MORBID ANATOMY.

In a nephritis of this type we should expect that the inflammatory products, the serum, white and red blood-cells, and coagulable matter from the blood-plasma would collect in the Malpighian bodies and tubes or infiltrate the stroma between the tubes, and that of the inflammatory products in the tubes and Malpighian bodies, a part would be discharged with the urine and a part be found in the kidney after death. We should also expect that the quantity of inflammatory products would be in proportion to the severity of the inflammation, and that an excessive number of pus-cells would belong to the especially severe forms of the disease. Still further, it is evident that with the milder examples of nephritis, with but little exudation, no inflammatory products might be found in the kidney after death, all having been discharged into the urine during life.

As a matter of fact, the kidneys do present just such changes.

In the mild cases we find no decided lesions in the kidney after death.

In the more severe cases the kidneys are increased in size, their surfaces are smooth, the cortical portion is thick and white, or white mottled with red, or the entire kidney is intensely congested. If the stroma is infiltrated with serum, the kidney is succulent and wet; if the number of pus-cells is very great, there will be little whitish foci in the cortex.

In such kidneys we find the evidence of exudative inflammation in the tubes, the stroma, and the glomeruli, all the changes being most marked in the cortical portion of the kidney.

The epithelium of the convoluted tubes is often simply flattened. As this same appearance is also found in the chronic congestion of heart disease, it seems probable that this change of the shape of the cells is merely due to the inflammatory congestion.

In other cases, not only is the epithelium flattened, but there is also a real dilatation of the cortex tubes. This dilatation involves groups of tubes or all the cortex tubes uniformly.

In other cases the epithelium of the convoluted tubes is swollen, opaque, degenerated, and detached from the tubes.

The tubes, whether with flattened epithelium or dilated, may be empty. More frequently, however, they contain coagulated matter in the form of irregular masses and of hyaline cylinders. The irregular masses are found principally in the convoluted tubes; they seem to be formed by a coagulation of substances contained in the exuded blood-plasma, and are not to be confounded with the hyaline globules so often found in normal convoluted tubes. The cylinders are more numerous in the straight tubes, but are also found in the convoluted tubes. They also are evidently formed of matter coagulated from the exuded blood-plasma, and are identical with the casts found in the urine.

The tubes may also contain red and white blood-cells.

In the cases in which there is an excessive emigration of white blood-cells, we find these cells in the tubes, in the stroma, or distending the capillary veins. This excessive emigration is not necessarily attended with exudation of the blood serum, and so the urine of these patients may contain no albumin. The white blood-cells are not usually found equally diffused through the kidneys, but are collected in foci in the cortex. These foci may be very minute or may attain a considerable size.

The glomeruli regularly are changed. The cavities of the capsules may contain coagulated matter and white and red blood-cells, just as do the tubes. The capsular epithelium may be swollen, sometimes so much so as to resemble the tubular epithelium, and this change is most marked in the capsular epithelium near the entrance of the tubes.

The most noticeable change, however, is in the capillary tufts of the glomeruli. These capillaries are normally covered on their outer surface by flat, nucleated cells, so that the tuft is not made up of naked capillaries, but each separate capillary throughout its entire length is covered over with these cells. There are also flat cells which line the inner surfaces of the capillaries, although not uniformly, as is the case in capillaries in other parts of the body. Still, in spite of the presence of all these cells, the outlines of the walls of the capillaries are fairly distinct.

In exudative nephritis the swelling and growth of cells on and in the capillaries change the appearance of the glomeruli. They are larger, more opaque, the outlines of the main divisions of the tuft are visible, but those of the individual capillaries are lost.

It is difficult to tell how much these changes in the glomeruli interfere with the passage of the blood through their capillaries.

In most cases of exudative nephritis the patients recover, and the glomeruli return to their natural condition.

In some examples of exudative nephritis we also find a thickening of the walls of the branches of the renal artery within the kidney. This thickening is principally due to a swelling of the muscle-cells in the walls of these vessels.

All these changes in the kidneys are of such a character that they are not likely to be followed by a chronic nephritis. On the contrary, after the patients have recovered, the kidneys return to their normal condition.

#### SYMPTOMS.

1. There are cases of acute nephritis of so mild a character that they may easily be overlooked. I think that these mild cases occur more frequently than is commonly supposed. The patients are hardly sick enough to go to bed. They have a little

headache, perhaps some aching in the back and limbs, loss of appetite, a little nausea, and the feeling of general malaise. They often think that they have taken cold. These indefinite symptoms last for one or two weeks, disappear, and the patient is well again. If the urine is not examined it is not known that the patient has been suffering from a mild nephritis. If the urine is examined it is found that the quantity is somewhat diminished, the specific gravity is not lowered, an appreciable quantity of albumin is present, with hyaline, granular, and epithelial casts, sometimes with red and white blood-cells. If the number of blood-cells is sufficient to color the urine, the patient's attention is attracted by the change in color; the diminished quantity he is apt not to notice. These changes in the urine last for four or five weeks and then disappear.

2. The ordinary cases of acute exudative nephritis vary indeed in their severity, but all give characteristic symptoms. The only diseases with which they can be confounded are : acute productive nephritis and exacerbations of a chronic nephritis. If any person seems to have several attacks of acute nephritis, it regularly means that he has a chronic nephritis with exacerbations.

The quantity of urine is diminished at the onset of the nephritis, and continues small until the activity of the inflammation has subsided ; then the quantity increases from day to day and may even exceed the normal. The quantity of the urine must be in proportion to the quantity of blood which passes through the kidneys, so that this quantity gives us a measure of the intensity of the congestion which is arresting the circulation of the blood through the kidney. Complete suppression of urine is a serious symptom, both because it denotes an intense nephritis and because it is of itself a cause of death. The production of only a few ounces of urine in each twenty-four hours is the rule in a great number of cases, and is not necessarily of serious import. If it only lasts a few days the patients do perfectly well. If the scanty excretion of urine is kept up for a number of days, opinions vary as to the results. Some believe that the diminished quantity of urine is the cause of the dropsy. Some think that the deficient excretion of excrementitious substances causes the convulsions. Some believe that the principal effect of a diminished excretion of urine is to cause bodily feebleness.

Unquestionably, the production of urine may be very small

for a number of days and yet the patients do well. Dr. Whitelaw (*Lancet*, September 29, 1877) reports a case of anuria, lasting for twenty-five days. The patient was a boy eight years of age. The suppression of urine began twelve weeks after the beginning of a scarlet fever. With the exception of two ounces passed on the thirteenth day, there was complete anuria for twenty-five days. Except for slight headaches and later slight œdema, there were no uræmic or dropsical symptoms throughout. There was no albuminuria and no fever. The boy was watched night and day. He recovered entirely.

The specific gravity of the urine remains normal or is higher while the quantity is small; when the quantity is increased the specific gravity falls a little.

The appearance of the urine is turbid, or smoke-colored, or bloody.

Albumin is present in very large quantities. Casts are numerous—hyaline, granular, nucleated, epithelial, and blood. There are also red and white blood-cells, and epithelial cells from the kidneys and from the bladder. As a rule the quantity of albumin and the number of casts are in proportion to the severity of the nephritis, but this is not always the case. Large quantities of albumin, numerous casts, and many red and white blood-cells may be found in the urine of kidneys which, after death, show no structural changes except in the glomeruli. While, on the other hand, small quantities of albumin and a few hyaline casts are compatible with a severe nephritis. Still further, the number of casts found in the urine during life is not always in proportion to the number of casts found in the corresponding kidneys after death.

The characteristic symptoms of acute exudative nephritis are: a febrile movement, with more or less prostration; headache, stupor, sleeplessness, restlessness, muscular twitching, general convulsions; dyspnœa, loss of appetite, nausea and vomiting; a pulse of high tension with exaggerated heart action, or hypertrophy of the left ventricle; dropsy and anæmia.

When acute nephritis complicates scarlet fever or one of the other infectious diseases, the patient may already have a febrile movement belonging to the primary disease. If the nephritis is primary, or if it is not developed until the fever belonging to the original disease has subsided, there is a rise of temperature belonging to the nephritis. This fever is in proportion to the

severity of the nephritis, and in children is sometimes as high as 105° F. The fever, however, does not usually continue more than a week, although the nephritis lasts longer.

Headache, restlessness, sleeplessness, delirium, and stupor during the first days of an acute nephritis seem to be of the same nature as they are in so many severe inflammations attended with fever. But later in the disease, after the temperature has fallen, they apparently depend upon the increased tension in the arteries. In the cases of prolonged anuria, however, there is a condition of mild delirium and stupor with a soft pulse.

General convulsions are of not uncommon occurrence, especially in children. They do not usually occur until after the nephritis has existed for several days. They are often preceded by involuntary contractions of groups of muscles. They may be preceded and followed by stupor. The frequency of their occurrence does not seem to be in direct relation to the quantity of urine excreted. They may be absent in fatal anuria, and present when the quantity of urine is nearly normal. It is the rule before and during the convulsions to have a marked increase in the tension of the pulse. While general convulsions are an alarming symptom, yet a great many children make a very good recovery after having them.

Loss of appetite, nausea, and vomiting at the beginning of the nephritis seem to be due to the febrile movement. Later in the disease it is probable that they are due to the disturbance of the function of the kidneys.

A pulse of high tension, exaggerated contractions of the heart, and sometimes hypertrophy of the left ventricle are present in some of the cases, not by any means in all of them. This disturbance of the circulation is evidently caused by contraction of the arteries. That the contraction of the arteries is due to the presence of irritating substances in the blood is probable, but not certain.

Dropsy is present in many of the cases. It is usually confined to the subcutaneous connective tissue. Its probable causes have already been discussed.

Anæmia, with a pallor of the skin out of proportion to the diminution in the quantity of hæmoglobin, is very often seen. We are still ignorant as to the way in which an acute nephritis causes such changes in the composition of the blood.

3. Acute exudative nephritis with an excessive production of



pus-cells. This is not to be confounded with embolic nephritis, nor with nephritis caused by cystitis. It is only a severe variety of acute exudative nephritis. It is seen both in children and in adults. I have seen it with scarlatina, diphtheria, and measles, and occurring without discoverable cause.

The invasion is sudden, with a high temperature and marked prostration. Restlessness, headache, delirium, and stupor are soon developed and continue throughout the disease. The patients rapidly lose flesh and strength and pass into the typhoid state. Dropsy is slight or absent altogether. The urine is not so much diminished in quantity as one would expect. Its specific gravity is not changed. Albumin, casts, and red and white blood-cells are present in considerable quantities, but not always early in the disease, and they may even be absent altogether.

Although this form of nephritis is not of common occurrence, the unusual characters of the symptoms and the great mortality are reasons for calling special attention to it. It differs from the ordinary form of exudative nephritis in that it behaves like an infectious inflammation, and that, although the emigration of white blood-cells is large, the exudation of serum may be small, and so the urine may show but little changes. It is probable that the nephritis is the result of some obscure form of bacteritic infection.

The ordinary duration of an acute exudative nephritis, which terminates favorably, is about four weeks, but may extend to eight. The recovery is a complete one, and there is no danger that chronic nephritis will follow.

#### PROGNOSIS.

The patients, who for three or four weeks have only the fever, prostration, loss of appetite, nausea, anæmia, dropsy, and changes in the urine, as a rule recover completely and are not at any time in real danger.

The development of the cerebral symptoms—the stupor, headache, sleeplessness, restlessness, and general convulsions—always causes anxiety, but yet even of these patients the larger number get entirely well. The cases with an excessive production of pus-cells differ from all the other forms of acute exudative nephritis, and are very fatal.

## TREATMENT.

We have to treat an acute exudative inflammation of the kidneys, which naturally runs its course in four weeks and terminates in recovery. We have also to treat the symptoms of this nephritis—the scanty urine, dropsy, vomiting, anæmia, and cerebral symptoms. We have to treat these conditions more frequently in children than in adults, and very often as complicating an infectious disease.

The most efficient treatment of the nephritis is the application of heat to the entire surface of the body. This can be done in a number of ways, but the best way is to wrap the entire body in a blanket wrung out of hot water. Such a hot pack can be used for an hour at a time once or twice a day. Of drugs the most reliable is aconite—one or two minims of the tincture every hour. It may be necessary to precede the aconite by giving one drachm of sulphate of magnesia every hour until the bowels move, or until eight doses have been taken. There are cases, in which the nephritis is not of very acute type, where digitalis seems to exert a favorable effect on the circulation. The preferable form of the drug is digitalin in doses of one one-hundredth of a grain.

The scanty urine often causes anxiety. Of course it is better that the patients should pass a fair quantity of urine, but I think that there is a tendency to exaggerate the dangers of scanty urine and to be too energetic in giving diuretics. As the diminution in the quantity of urine is due to the congestion of the kidneys, if we can decrease the congestion the urine will increase. The best way to do this is to apply heat to the surface of the body. The use of diuretics is to be avoided. The attempts to make up for the scanty production of urine by purging or sweating the patient have never seemed to me to be of any practical use.

The febrile movement in an acute nephritis requires no treatment.

The prostration, loss of appetite, nausea, and vomiting only call for rest in bed and a fluid diet.

The anæmia ought to be prevented or relieved, but while the nephritis is still active I know of no way in which this can be done. When convalescence is established, then the anæmia readily improves with the ordinary methods of treatment.

The dropsy is subcutaneous, and even when considerable, does little harm. It disappears of itself as the nephritis subsides. The rest in bed and the hot pack are all the treatment necessary for it. To give diuretics or cathartics to get rid of the dropsy is quite useless.

The cerebral symptoms are the ones to which most attention has been directed. There can be no question that they accompany a contraction of the arteries with increased arterial tension and labored action of the heart. No matter what views one may entertain as to the cause of this change in the circulation, I believe that treatment is best directed to the arteries themselves, rather than to the uncertain causes of their contraction. Fortunately there are drugs which stop contraction of the arteries promptly and efficiently. Of these drugs the most suitable are : aconite, nitro-glycerin, chloral hydrate, and opium, preferably given in small doses and at regular intervals before the cerebral symptoms are marked, but in large doses hypodermically or by the rectum to stop a severe attack.

It is wise to watch the condition of the heart and arteries, and as soon as increased arterial tension is developed, not to wait for the manifestation of the cerebral symptoms, but to try to relieve it at once.

The way in which we manage the patients, therefore, is as follows : They are put to bed or kept in the house until the nephritis has run its course. They are put on a fluid diet, preferably milk, and the skin of the entire body is cleaned once a day. For many cases no other treatment is necessary.

If vomiting is troublesome it can usually be controlled by adding oxalate of cerium and bicarbonate of soda to the milk. For the restlessness and sleeplessness chloral hydrate, the bromides, or opium may be employed.

If the nephritis is of severe type the patient is wrapped in a blanket wrung out of hot water and kept in it for one hour either once or twice every day. In addition we give one drachm of sulphate of magnesia every hour until the patient has taken eight doses or the bowels begin to move. This is followed by one or two minims of tincture of aconite every hour.

Throughout the disease we watch the pulse, and as soon as it shows any increased tension give chloral hydrate in doses of from two to five grains every three hours.

If severe headache, muscular twitchings, or general convul-

sions occur, to most of the patients we give chloral hydrate in doses of from 5 to 20 grains by the rectum, or nitro-glycerin in doses of from  $\frac{1}{200}$  to  $\frac{1}{50}$  of a grain hypodermically, or morphine in doses of from  $\frac{1}{10}$  to  $\frac{1}{6}$  of a grain hypodermically. In strong and robust adults with a good deal of venous congestion, general blood-letting may be advisable. For the relief of the convulsions urethane in solution, given in repeated doses up to 100 grains in twenty-four hours, is said to be of service.

As the nephritis subsides the milk is gradually replaced by solid food, and iron and oxygen are given.

### ACUTE PRODUCTIVE (OR DIFFUSE) NEPHRITIS.

#### DEFINITION.

An acute inflammation of the kidneys, characterized by exudation from the blood vessels, a growth of new connective tissue in the stroma, and changes in the epithelium and the glomeruli.

*Synonyms.*—Acute Bright's Disease ; Parenchymatous Nephritis ; Croupous Nephritis ; Glomerulo-nephritis.

#### ETIOLOGY.

This is the most serious and important of the forms of acute nephritis, for the reason that its lesions are from the first of a permanent character. It does not follow exudative nephritis, nor is it merely a modification of it; from the very outset it is a different form of inflammation. In the kidneys of persons who have been sick only a few days, the characteristic lesions are already evident. Productive nephritis is governed by the same law as that which belongs to productive inflammation in other parts of the body—the disposition of the inflammation to continue as a subacute and chronic condition. It is of importance to recognize that in exudative nephritis the lesions are temporary, and after their subsidence the kidneys return to their normal condition, just as the lungs do after a lobar pneumonia. In productive nephritis, on the other hand, some of the lesions are permanent, the kidneys can never return to their normal condition, just as in an interstitial pneumonia the lung never gets rid of the new connective tissue.

Post-scarlatinal nephritis is nearly always of the productive form. Nephritis complicating diphtheria or developed during

pregnancy is very frequently of this type. A primary nephritis in a person over twelve years old, if of subacute form, is almost invariably a productive nephritis. On the other hand, this form of nephritis very seldom complicates any of the infectious diseases except scarlatina and diphtheria.

These facts assist very much in making the diagnosis between the two forms of acute nephritis. It is easy to remember that post-scarlatinal nephritis and primary nephritis of subacute type are nearly always of the productive form; and that nephritis with diphtheria and pregnancy is often of the productive form; while acute nephritis under all other conditions is regularly of the exudative form.

#### MORBID ANATOMY.

The kidneys are increased in size, the capsules are not adherent, the surfaces are smooth. The cortical portion is red, or white, or mottled. The mucous membrane of the pelvis is sometimes congested. Of the tubules in the cortex, in some the epithelium is flattened, in some there is coagulated matter or casts, in some the epithelium is swollen, degenerated, or contains globules of fat. In those parts of the cortex where there is a growth of new connective tissue, the tubes may be atrophied. The tubules of the pyramids show but little change except that they may contain casts. In the stroma of the cortex there is a growth of new connective tissue, varying in different kidneys as to the relative proportion of cells and basement substance. This new tissue in many of the kidneys follows the line of the arteries which run up into the cortex, so that it takes the form of wedges. But in other kidneys the new tissue is diffuse, or in irregular patches.

Many of the glomeruli show only an increase in the size and number of the cells which cover the capillaries, with some swelling of the capsule cells. But in others there is an extensive new-growth of capsule cells which compresses the tuft of vessels. This growth of new cells from the capsule cells must not be confounded with accumulations of white blood-cells within the capsules, nor with the growth of new cells on the walls of the capillaries. The glomeruli which are changed in this way are in groups, each group corresponding to some one artery.

The whole picture of the nephritis is that of a combination of exudative and productive inflammation.

When such a nephritis becomes chronic it is often possible to follow its course for many years, and to see at the end of that time that the anatomical changes in the kidney are of the same kind, but much more extensive.

#### SYMPTOMS.

Of the patients who suffer from this form of nephritis, a certain number behave as if they had a simple exudative nephritis. There is a rise of temperature with more or less prostration. Cerebral symptoms are marked—headache, stupor, sleeplessness, restlessness, muscular twitchings, and general convulsions. The arteries are contracted, the pulse is of high tension, the heart's action is exaggerated, the left ventricle may be hypertrophied, there is dyspnœa. The appetite is lost, there may be nausea and vomiting. The urine is scanty or suppressed, it is colored by blood, and contains much albumin and many casts. The patients are very sick and much more likely to die than they are with an exudative nephritis.

Such a nephritis may, however, apparently run its course. At the end of four weeks the symptoms subside and the patients get better. They may then remain in ordinary health without renal symptoms for weeks, months, or years. But sooner or later they have another acute attack, or they gradually develop the symptoms of a chronic nephritis.

The more ordinary cases have a gradual invasion, and run a subacute rather than an acute course.

In some of the patients at first there are only loss of appetite, headaches, and an increasing pallor of the skin and mucous membranes; the dropsy does not come on until after many days.

In some of the patients dropsy of the legs is the first and, for a time, the only symptom. They continue to eat well, feel well, and attend to their work.

In most of the patients dropsy of the legs and face, anæmia, headache, sleeplessness, loss of appetite, nausea and vomiting are developed at about the same time.

The urine is only moderately diminished in quantity; it often contains no blood, there is a large quantity of albumin and a considerable number of casts. The specific gravity remains normal, or falls a little.

The cases vary a good deal as to their severity

Some of the patients are not at any time very sick. A moderate subcutaneous œdema, anæmia, headache, and disturbances of digestion last for a few weeks, then disappear, and the patients seem to be well. Some of them do get well, but the majority either have other attacks of the same character, or develop the symptoms of chronic nephritis. It is surprising for how many years some of these patients go on in apparent good health, although the kidneys are really becoming more and more diseased.

In some patients the dropsy is much more extensive and involves the serous cavities as well as the subcutaneous tissue. For a number of weeks these patients are in bed and very badly off. And yet even the bad attacks may subside altogether, the patients are apparently well, are able to go back to their work, and have no more trouble for years.

In some patients there is first a well-marked attack of dropsy, anæmia, headache, sleeplessness, loss of appetite, nausea and vomiting, which lasts for a few weeks. Then the symptoms subside and the patients are pretty well, but not very well. After this they have attacks of the same kind at intervals of weeks or months, and this may go on for years. In hospital patients the attacks regularly come on every winter, and the patients are comparatively well in the summer. Each attack, however, is worse than the preceding, and finally there comes an attack which proves fatal. In these long cases the specific gravity of the urine becomes lower from year to year.

The severe and progressive cases are most distressing to witness. The patients are constantly getting worse, and yet months may elapse before their sufferings are terminated by death. The color of the skin and of the mucous membranes becomes more and more white; headaches are constant and troublesome; sleep is difficult and unrefreshing; the eyesight is impaired or lost altogether; there is no appetite, but rather constant nausea and irritability of the stomach; from time to time the arteries are contracted and there is a disposition to muscular twitchings and general convulsions. The dropsy constantly increases, no matter how large the excretion of urine. The subcutaneous connective tissue is everywhere œdematous and the serous cavities are filled with serum. It seems as if the blood serum was unable to remain in the vessels, it escapes everywhere.

## PROGNOSIS.

The majority of cases of acute productive nephritis terminate unfavorably. Either the disease goes on continuously and the patients die at the end of a few days or a few months; or the acute symptoms subside and a chronic nephritis supervenes. It is not wise, however, to give too unfavorable a prognosis even in severe cases; great improvement and even complete recovery are possible. I see from time to time persons in apparently good health and able to earn their living, concerning whom I have given a very unfavorable prognosis many years ago.

## TREATMENT.

In those cases in which the disease behaves like an acute exudative nephritis the indications for treatment are the same as in the latter disease, although the results are not as satisfactory.

The subacute cases have to be managed differently. At first it is wise to keep the patients in bed and on an exclusively milk diet. In some of the patients the daily use of the hot pack seems to be of service; in some nothing is gained by its use; in some it is, I think, harmful. I do not know how to distinguish the appropriate cases for the hot pack except by trying its use for a few days.

Digitalis, preferably in the form of digitalin in doses of  $\frac{1}{100}$  of a grain, seems in some of the cases to exert a favorable effect on the nephritis. At all events the quantity of the albumin in the urine diminishes and the patients improve; but in some cases it does nothing. In the same way morphine in small doses, sometimes not more than  $\frac{1}{80}$  of a grain, relieves the headache, sleeplessness, and nausea, and the patients are evidently better for it. But there are other patients to whom the morphine is of no service whatever.

The dropsy is always of consequence. It is associated with a soft pulse; a faint heart action, rather feeble than forcible; no great disposition to venous congestion. The composition of the blood is profoundly changed by the diminution in the quantity of hæmoglobin and the number of red blood-cells, and probably in other ways which we do not appreciate. The quantity of urine may be either diminished or increased. The dropsy, there-



fore, does not seem to depend on changes in the blood pressure or in the quantity of urine, but rather on changes in the composition of the blood or in the walls of the arteries. It is a dropsy which it is very difficult to treat intelligently.

There are cases in which the dropsy will disappear simply with the rest in bed and the milk diet.

There are cases in which profuse sweating by the hot-air bath or the hot pack diminishes the dropsy. But some of these patients cannot be made to sweat, some of them are too much depressed by the heat, in some the sweating does not diminish the dropsy.

Hydragogue cathartics, such as jalap and elaterium, will often diminish the dropsy for a time. Their use, however, cannot be continued for any length of time without irritating the stomach and intestines.

The daily use of good massage with compression of the legs by bandages is sometimes of real value.

Digitalis, caffeine, and strophanthus in many cases act efficiently, even when they do not increase the quantity of urine. They are the most useful drugs for this particular purpose.

The use of diuretics is limited to the cases in which the quantity of urine is diminished. In a patient with increasing dropsy, who is already passing 90 or 100 ounces of urine a day, diuretics are not indicated. The drugs ordinarily employed to effect diuresis are those which act on the circulation—digitalis, caffeine, strophanthus, and convallaria; and those which are supposed to act on the kidneys—acetate of potash, lactate of strontium, squills, diuretin. Good results are reported from the use of those drugs. But experience shows that in some patients the quantity of urine cannot be increased, and in others the increase in the quantity of urine is not followed by diminution of the dropsy.

It would seem as if the disposition to dropsy could be controlled if we could control the composition of the blood, increase the number of the blood-cells, and raise the specific gravity of the blood serum. Unfortunately we do not know how to do this.

When necessary, to make the patient more comfortable, we have to tap the peritoneal and pleural cavities and to puncture the skin of the legs.

If the patients improve, the milk diet is to be gradually replaced by solid food, iron is to be given in fair doses, and the

patient gets out of bed and out of the house. At this time climate becomes a matter of much importance. The patient should be sent to a warm, dry, equable climate, where he can lead an out-door life.

### CHRONIC PRODUCTIVE (OR DIFFUSE) NEPHRITIS WITH EXUDATION.

#### DEFINITION.

A chronic inflammation of the kidney attended with a growth of new connective tissue in the stroma, permanent changes in the glomeruli, degeneration of the renal epithelium, exudation from the blood-vessels, and sometimes changes in the walls of the arteries.

*Synonyms.*—Chronic Bright's Disease ; Chronic Parenchymatous Nephritis ; Chronic Glomerulo-nephritis ; Waxy Kidney ; Large White Kidney ; Chronic Diffuse Nephritis ; Chronic Desquamative Nephritis.

It has been customary to hold that in all these kidneys the primary and most important changes are in the renal epithelium, while in another set of kidneys the primary and important changes are in the stroma. In other words, that the cases of chronic nephritis can be divided into two classes—parenchymatous nephritis and interstitial nephritis.

I do not think that this classification is supported by facts.

In all the forms of chronic nephritis changes are to be found in the renal epithelium, the glomeruli, and the stroma. Whether the changes in the stroma, the glomeruli, or the epithelium are the more marked makes no difference in the clinical symptoms. But the presence or absence of exudation from the renal blood-vessels does correspond to a marked difference in the symptoms. The existence of the exudation from the renal vessels is easily shown by the presence of serum albumin in the urine. In this way we readily distinguish two forms of chronic nephritis, one with exudation and one without.

The way of looking at the matter, then, is this :

We find after death from chronic nephritis a great many varieties in the gross appearance of the kidneys. Some are large, some are small, some are red, some are white, etc. There is no regular correspondence between these different gross appearances of the kidneys and the clinical symptoms.

We find in these same kidneys changes in the renal epithelium in the stroma, in the glomeruli, and in the arteries. Sometimes one, sometimes the other of these elements of the kidneys is the most changed. There is no regular correspondence between the predominance of the changes in one of the kidney elements over the other and the clinical symptoms.

The easiest working scheme is to admit that in chronic nephritis all the elements of the kidney are more or less changed, but that the cases vary as to whether there is or is not an exudation of serum from the blood-vessels. The presence or absence of such an exudation does correspond to a well-marked difference in the clinical symptoms.

In the present state of our knowledge and for clinical purposes, we divide all the cases of nephritis into two classes, chronic nephritis with exudation and chronic nephritis without exudation.

It is admitted that it is easy to divide up these kidneys, according to their anatomical changes, into a number of fairly well-marked classes. But as this division does not correspond to clinical divisions it is valueless for clinical purposes.

Although it is convenient to describe two forms of chronic nephritis—one with much albuminuria and dropsy, and one with little or no albuminuria or dropsy—yet it must be remembered that these are not separate lesions of the kidneys, but varieties of the same lesion. For in all these kidneys two changes are constant—productive inflammation of the glomeruli and stroma, and degeneration of the renal epithelium. The only real difference between the kidneys is whether, beside the growth of new tissue and degeneration of renal epithelium, there is or is not an exudation of serum from the blood-vessels of the kidneys.

In speaking of the exudation of serum from the vessels and its presence in the urine, we speak of it as it occurs during the whole course of the disease, and not as it occurs for short periods. We mean that in an exudative chronic nephritis there is usually a large quantity of albumin in the urine, but that there may be periods during which the albumin diminishes or entirely disappears. In the same way, in a non-exudative nephritis there may be periods during which albumin is present in considerable quantities. Generally speaking, the character of the clinical symptoms will vary with the presence or absence of the albumin.

## ETIOLOGY.

A considerable number of cases of chronic nephritis follow an attack of acute or subacute productive nephritis. The conditions of chronic congestion and chronic degeneration of the kidney are not infrequently followed by a true nephritis.

Syphilis, chronic tubercular inflammation of any part of the body, chronic endocarditis, and chronic suppurative inflammations are often complicated with chronic nephritis.

It is very difficult to find a satisfactory cause for the primary cases. There are many of these, especially in young and middle-aged adults. The nephritis is developed in a slow, insidious way in persons whose previous health had been good, and in whom no exciting cause is discoverable.

## MORBID ANATOMY.

*Gross Appearance of the Kidney.*—There is considerable variety in the gross appearance of the kidneys. The types which I have seen most frequently are as follows :

1. Large white kidneys, weighing together sixteen ounces or more, the capsule adherent or not, the surface smooth or nodular, the cortex thick and white, the pyramids large and red.
2. Large mottled kidneys. These resemble the large white kidneys in every respect except that the cortex, instead of being white, is mottled in a variety of ways with white, yellow, red, and gray.
3. Kidneys which resemble types one and two, but are not enlarged, the kidneys together not weighing over nine ounces.

The majority of the kidneys in chronic nephritis follow these three types.

4. Small kidneys, weighing together not more than five ounces, the capsules adherent or not, the surfaces nodular, the cortex thin, atrophied, white, the pyramids rather large and red. These kidneys belong to persons who have had symptoms of kidney disease for many years, with periods of apparent recovery.

5. Kidneys which have the ordinary appearance and consistence of the chronic congestion due to heart disease, but in addition the capsules are adherent and the surfaces finely nodular.

6. Kidneys of different sizes—large, medium-sized, and small,

with adherent capsules and nodular surfaces. The cortex is gray, or gray mottled with red. The kidneys do not look at all like the large white kidneys. This is a type of frequent occurrence.

7. Kidneys which in their size, color, and general appearance are hardly to be distinguished from normal kidneys, except that their capsules are adherent.

8. Kidneys of small size, weighing together not more than four ounces, with adherent capsules. The cortex is atrophied red, and irregular. These kidneys are found in persons who have given symptoms of kidney disease for a number of years.

It might naturally be supposed that such marked differences in the gross appearance of the kidneys would correspond to equally marked differences in the clinical histories and minute lesions. This, however, is not the case. The clinical histories are practically interchangeable, and the minute lesions are essentially the same.

*Microscopical Appearances.*—If we make vertical sections of the cortex of all these kidneys, no matter what their size or color, we get with a low magnifying power the same general picture. Instead of the uniform and orderly arrangement of tubes and glomeruli which we see in the normal kidney, the tubes seem to be obliterated in some places and dilated in others. There is a growth of fibro-cellular tissue in regular wedges, in irregular patches, or diffuse between the tubules.

If we examine the different constituents of the kidney in detail we find :

*The tubes* are in some places of normal size, in some places atrophied, in some places dilated. The atrophied tubes are in the patches of new connective tissue. The dilated tubes are not very large, nor do they form cysts.

The epithelium of the tubes is in some places merely flattened. These tubes are empty, or contain coagulated matter, casts, and red and white blood-cells. In other tubes the epithelium is more or less swollen, sometimes so much so as to completely fill the tubes. In still other tubes the epithelial cells are swollen, their reticulum is very coarse with large meshes, and they are infiltrated with fat. The kidneys vary as to which of these changes in the epithelium predominates, but all of them may be found in the same kidney.

The new connective tissue is in the form of wedge-shaped

masses in the cortex which follow the line of the straight arteries and veins, or it is in irregular masses, or it is arranged diffusely so as to separate the tubes from each other. The longer the nephritis lasts, the greater is the quantity of new connective tissue. The relative proportion of basement substance and cells and the density of the basement substance vary in the different kidneys. The new tissue is well supplied with blood-vessels.

*The glomeruli* are changed in several different ways :

1. They resemble the glomeruli in acute exudative nephritis. They are large, the convolutions of the capillaries are seen with difficulty, there is a very great increase in the number of the cells which cover the capillaries, but these new cells are not of large size. We also see glomeruli, which apparently have been of this type, small and atrophied.

2. There is an increase not only in the number, but also in the size, of the cells which cover the capillaries. These cells are so large that they project outward from the surface of the glomerulus. There is also an increase in the size and number of the cells within the capillaries. These glomeruli are found in all stages of atrophy.

3. The capillaries are changed in the same way by a growth of large cells on their outer surfaces and within them. In addition there is a very extensive cell-growth beginning in the cells which line the capsule. The mass of new cells produced in this way may be so great as to compress the capillaries. The glomeruli also become atrophied, the capillaries are shrunk, and the capsule cells changed into connective tissue.

4. If chronic congestion of the kidneys is followed by chronic nephritis, the dilatation of the capillaries due to the congestion continues, and there is added an increase in the size and number of the cells which cover the capillaries.

5. The walls of the capillaries are the seat of waxy degeneration, while the cells which cover them are increased in size and number.

6. Besides the atrophied glomeruli already described, there are others which are small and shrunk, with comparatively little new growth of cells.

*The arteries* are not infrequently much altered by inflammatory changes. There is a growth of cells and basement substances from the inner surface of the artery which obstructs its lumen ; or there is a thickening of each of the three coats of the artery ;

or all the coats of the artery are thickened and converted into a uniform mass of dense connective tissue; or the wall of the artery undergoes waxy degeneration.

#### SYMPTOMS.

*The urine* varies in quantity at different times in the course of the disease. In the earlier periods the urine is often scanty or even suppressed. If the disease goes on rapidly the quantity of urine may continue small; if it goes on slowly the quantity is often increased. In some of the worst cases, with general dropsy, the patients will pass more than one hundred ounces of urine a day.

The specific gravity and the proportion of urea to the ounce of urine slowly diminish as the disease progresses. This is the rule, but there are exceptions to it. In cases which improve the quantity of urea, after being much diminished, may increase until the patient excretes the full normal quantity for the twenty-four hours.

In the cases of shorter duration the specific gravity is apt to run between 1.012 and 1.020. In the very chronic cases it will be between 1.001 and 1.005. A very low specific gravity indicates a large growth of connective tissue in the stroma of the cortex, or waxy degeneration of the capillaries of the glomeruli and of the arteries of the kidney. Many persons who think that they have kidney disease get into the habit of drinking large quantities of mineral waters. This, of course, gives them urine of low specific gravity. In all doubtful cases it is necessary to determine the quantity of the whole excretion of urea for the twenty-four hours. There are patients in whom the quantity of urea is the principal factor in enabling one to decide between albuminuria without nephritis and chronic nephritis with exudation.

The urine regularly contains albumin and casts. During the active periods of the disease the quantity of albumin is very large; in the slow, prolonged cases the quantity is much smaller, and at times it may disappear altogether. Generally speaking, with large quantities of albumin the patients are dropsical and anæmic; with small quantities of albumin they are anæmic but not dropsical. There seems to be a common cause for the exudation of serum from the blood-vessels throughout the entire body, the serum infiltrating the tissues, accumulating in the serous cavities, and mixing with the urine.

The number of casts is regularly in proportion to the quantity of albumin, but there are exceptions to this rule.

A peculiar pallor of the skin and white color of the sclerotic is seldom absent. This gives to the patient a face very characteristic of chronic nephritis. In making a diagnosis in doubtful cases a good deal of importance is to be attached to the presence or absence of this appearance of the face. The change in the color corresponds to a diminution in the quantity of hæmoglobin and in the number of red blood-cells. These changes in the blood are often not far advanced, but sometimes they are, and some patients even die with the symptoms of pernicious anæmia.

*Dropsy* may be considered almost a constant symptom of chronic exudative nephritis. There is an infiltration of the subcutaneous connective tissue with serum and an accumulation of serum in the serous cavities. The position of the fluid varies with that of the patient, accumulating in the dependent portion of the body. There is much variety as to the extent of the dropsy. In some patients there is never anything more than a moderate œdema of the legs, while in others a marked general dropsy is the most prominent symptom of the disease. There is also a variety as to the time of appearance and the duration of the dropsy. It may be one of the first symptoms of the nephritis, or it may not occur until late in the disease. When it is once established it may never leave the patient, or it may appear and disappear at irregular intervals.

Many of the patients are troubled with headache and sleeplessness. In some of them these symptoms exist only when the pulse is of high tension and disappear when the pulse becomes soft. In others, however, the headache and sleeplessness persist with a soft pulse. It must not be forgotten that these symptoms may also depend on digestive disturbances, and not on the disease of the kidneys.

*Acute uræmic attacks* with contraction of the arteries, dyspnœa, vomiting, convulsions, etc., may occur at any time in the course of a chronic exudative nephritis. But they are of very much more frequent occurrence with the non-exudative form of the disease.

*Chronic uræmia*, on the contrary, is one of the ordinary ways in which an exudative nephritis proves fatal. The condition belongs to the later stages of the disease. It is developed rather



gradually, but when once established is permanent, not disappearing up to the time of the patient's death. The patients are in a condition of alternating delirium and stupor, with a rapid, feeble, soft pulse.

*Simple neuro-retinitis*, or nephritic retinitis, may be developed at any time in the disease. Both eyes are regularly involved. The impairment of vision may be very slight, or considerable, or the patients may become entirely blind. With such a neuro-retinitis the prognosis of the nephritis is especially bad.

*Dyspnœa* is a nearly constant symptom, but it is not always the same kind of dyspnœa, nor produced by the same causes. It may be due to hydrothorax, to œdema of the lungs, to contraction of the arteries, or to failure of the heart's action.

The dyspnœa due to contraction of the arteries is common to both forms of chronic nephritis. It may be developed at any time during the course of the disease. It comes on in attacks, especially at night and in the early morning, and is worse when the patients lie down. It often begins while the patient is apparently in good health, but is a sure premonition of serious disease.

In some cases these attacks of dyspnœa can be controlled and the patient kept apparently well for months and even years. But as the attacks are repeated, they are more severe and more stubborn. The heart's action fails in addition to the contraction of the arteries, and the dyspnœa becomes of such a character that it can only be relieved by death.

In other patients the first attack of dyspnœa is also the last. It cannot be relieved by any treatment, and continues up to the time of the patient's death.

*A catarrhal bronchitis* with cough and expectoration is sometimes an annoying symptom. The cough fatigues the patient, and it is difficult to control it.

*Loss of appetite, nausea and vomiting* are frequent symptoms. When they do not already exist it is easy to cause them by the use of improper drugs.

*The heart* is very often affected. The disease of the kidneys after a time produces hypertrophy of the left ventricle. This does no harm until the time comes when the heart's action fails in spite of the hypertrophy, then the dyspnœa and the dropsy follow.

Chronic endocarditis, chronic myocarditis, and dilatation of

the ventricles are associated with chronic nephritis in two ways : They may cause a chronic congestion or degeneration of the kidney which is afterward followed by a nephritis ; or the heart disease and kidney disease are developed in the same person, neither one of them secondary to the other. In these patients it is by no means easy to tell how much of the dropsy, the dyspnœa, and the loss of nutrition belongs to the heart disease and how much to the nephritis.

#### COURSE OF THE DISEASE.

There is hardly any limit to the variations of the disease, but the most constant symptoms are anæmia, dropsy, and albumin in the urine.

The following are some of the ordinary examples of this form of nephritis :

1. There are cases in which the symptoms are nearly continuous. The patients begin with anæmia, headache, disturbances of digestion, and a little dropsy. No one of these symptoms is at first very marked ; the patient is not in bed, he does not feel very sick, but even at this time the urine contains a large quantity of albumin. As the weeks and months go on all the symptoms grow worse : the anæmia is more profound, the headaches and disturbances of digestion more troublesome, the dropsy involves more of the subcutaneous tissue and the serous cavities, so that the patient becomes more and more helpless. There may be intercurrent attacks of acute uræmia with contraction of the arteries, or loss of eyesight, or a troublesome bronchitis. The ordinary duration of this form of the disease is from one to three years. The patients die with the most extreme dropsy, or in the state of chronic uræmia, or both these conditions exist together.

2. There are cases in which the anæmia, the dropsy, and the dyspnœa come on in attacks which last for weeks or months. Between the attacks the patients are comparatively well, often able to work, although the urine always contains albumin. These patients often go on for a number of years, better in the summer and worse in the winter. But each successive attack is more serious than the preceding, and finally there comes an attack from which the patient does not recover.

3. There are cases in which a number of years before death the patients have an attack of acute or subacute nephritis with

anæmia, dyspnœa, dropsy, albumin in the urine, and all the usual symptoms. From this they apparently recover completely and seem to be in their ordinary health. The urine continues to contain a little albumin, or on some days the albumin disappears altogether. From year to year the specific gravity slowly falls. The exudation from the blood-vessels of the kidneys stops, but the chronic productive inflammation of the kidneys continues. Finally, after exposure, with an accident, with a pneumonia, or without discoverable cause, all the symptoms of a subacute nephritis are rather suddenly developed, and the patient soon dies.

4. There are cases which for years have no symptoms except pallor of the skin and mucons membranes, and urine of low specific gravity which habitually contains a moderate quantity of albumin. These patients must not be confounded with cases of simple anæmia, nor with those of persistent albuminuria without kidney disease. The diagnosis is sometimes quite difficult. The prognosis in these patients depends upon the specific gravity of the urine. If it is constantly below 1.010 the prognosis is bad, no matter how well the patients may feel.

5. There are cases in which the first symptom is the attack of spasmodic dyspnœa. This may continue and other renal symptoms rapidly develop. More frequently, however, the dyspnœa is palliated or relieved by treatment. The patient then goes on for months or years with occasional attacks of dyspnœa, each one more severe and harder to control, until finally other renal symptoms appear.

6. There are the cases complicated with endocarditis, myocarditis, or dilated heart. In these patients we have the association of cardiac and renal symptoms, either one predominating.

7. There are cases in which all the symptoms disappear, and the urine returns to its natural condition. If this improvement continues for a number of years it seems probable that the nephritis has come to a standstill, and that enough kidney tissue has been left unimpaired to carry on the functions of the organ.

#### TREATMENT.

In the second and third groups of cases just described the attacks are of acute or subacute character. The conditions calling for treatment are :

The nephritis.  
 The albuminuria.  
 The dropsy.  
 The headache and sleeplessness.  
 The nausea and vomiting.  
 The contraction of the arteries.  
 The anæmia.  
 The dyspnœa.

For the *nephritis* the patient should be kept in bed and placed on a fluid diet. He should have a hot pack twice a day, once a day, or every other day, as he will bear it. The most useful drugs are : morphine in very small doses, digitalin gr.  $\frac{1}{100}$ , and aconite  $\mathfrak{M}$  1-3.

The quantity of *albumin* in the urine can be diminished by the hot pack and by the use of digitalin gr.  $\frac{1}{100}$  four times a day.

The *dropsy* is favorably affected by the hot pack. It can be sensibly diminished by cathartics, of which elaterium is perhaps the best. But the effect of cathartics is temporary, and their use cannot often be repeated.

For many patients digitalis in some form is the most useful drug for the dropsy, and it can be continued without injury for weeks and months. Favorable results are reported from the use of lactate of strontium in 30-grain doses four times a day, and from diuretin in 5-grain doses three times a day. In some patients caffeine, convallaria, or strophanthus will answer better than digitalis.

If there is much fluid in the serous cavities it should be drawn off with the aspirator. If the dropsy of the subcutaneous connective tissue is excessive the skin is to be pricked and the fluid allowed to drain off.

The *headache* and *sleeplessness* may exist with or without contraction of the arteries. If the arteries are not contracted, morphine in very small doses, codeine, or the bromides can be used. If the arteries are contracted morphine in larger doses and chloral are the best drugs.

The *nausea* and *vomiting* are controlled by the milk diet, or it may be necessary to add an alkali to the milk, or to use peptonized milk or kumyss.

*Contraction of the Arteries.*—The character of the radial pulse and the heart's action are to be constantly watched. We do not wait for the dyspnœa, or vomiting, or convulsions to make their

appearance, but as soon as the pulse shows an increased tension we begin with nitro-glycerin, chloral hydrate, morphine, or potassium iodide. It is to be remembered that morphine in considerable doses can only be given to patients with this form of nephritis when the arteries are contracted. At all other times it is very easy to have dangerous and even fatal results with any preparation of opium.

The *anæmia* of chronic nephritis does not behave like simple anæmia. Iron is not a specific for it, although it may be of service. The greatest improvement in the anæmia is effected by the subsidence of the nephritis.

The *dyspnoea* is dependent either upon contraction of the arteries or upon dropsy. The treatment for it, therefore, is either the treatment of dropsy or the treatment of contraction of the arteries.

In these two groups of cases treatment carried on in these ways is often very satisfactory. All the symptoms subside and disappear. But it must be remembered that the kidneys have become changed in their structure, that a chronic productive nephritis still continues, that relapses and exacerbations are to be expected.

As the symptoms subside the patient gets back to a solid diet, is out of bed, and then out of doors. If it is possible for him to spend the next two years in a warm climate where he can lead an out-of-door life, the probabilities of permanent improvement will be much greater.

In the first set of cases, those in which the symptoms continue and get steadily worse, treatment is very unsatisfactory. We try the measures that have just been described, but they are of no avail. We cannot even alleviate symptoms, the drugs do not help at all. After a time it becomes evident that there is no use in continuing plans of treatment which do nothing, and we employ very little treatment.

The fourth set of cases have no acute attacks, no great change in the general health, nothing but the anæmia and the changes in the urine. The management of these cases requires much judgment.

The patients should have a liberal and varied diet, and yet every form of indigestion is to be guarded against. They do best if they can live in a warm climate all the year round. But even in an unfavorable climate they need out-of-door exercise.

The drugs indicated are those for the relief of indigestion, and the preparations of iron.

The patients who begin with attacks of dyspnœa, without other renal symptoms, can often be relieved and enabled to work for a number of years. The dyspnœa is associated with a pulse of high tension; if we can relieve this the dyspnœa disappears. The best drugs for this purpose are nitro-glycerin, chloral hydrate, and potassium iodide.

In the cases with chronic endocarditis, myocarditis, or dilatation of the ventricles, the management of the heart's action becomes a matter of great importance.

### CHRONIC PRODUCTIVE NEPHRITIS WITHOUT EXUDATION.

#### DEFINITION.

A chronic inflammation of the kidney attended with a new growth of connective tissue in the stroma, permanent changes in the glomeruli, degeneration of the renal epithelium, and sometimes changes in the walls of the arteries.

*Synonyms.*—Chronic Bright's Disease; Cirrhosis of the Kidney; Granular Degeneration; Interstitial Nephritis; Chronic Indurative nephritis; The Arterio-sclerotic Kidney.

#### ETIOLOGY.

While this form of nephritis is especially common in persons over forty-five years old, it is by no means rare in young adults, and is occasionally seen in children.

It seems to be caused by chronic alcoholism, lead poisoning, gout, and by the same conditions as those which cause emphysema, endocarditis, and cirrhosis of the liver. It follows chronic congestion of the kidney, hydro-nephrosis, and chronic pyelitis.

#### MORBID ANATOMY.

*The Kidneys.*—The larger number of the affected organs are found after death to be diminished in size; the two kidneys together may not weigh more than two ounces. The capsules are adherent; the surfaces of the kidneys are roughened or nodular; the cortex is thin and of a red or gray color.

A considerable number of these kidneys, however, do not differ in their size or appearance from normal kidneys, except that their capsules are adherent and their surfaces roughened.

Occasionally the kidneys are large, weighing together from 16 to 32 ounces, with smooth or nodular surfaces, and a cortex of red, gray, or white color.

If the nephritis follows chronic congestion, the kidneys remain hard, but the cortex becomes thinned, the capsules adherent, and the surface roughened.

There is a growth of new connective tissue in the cortex and also in the pyramids, which becomes more and more extensive as the disease goes on. In the cortex the new tissue follows the distribution of the normal subcapsular areas of connective tissue, is in the form of irregular masses, or is distributed diffusely between the tubes. In the pyramids the growth of new connective tissue is diffuse.

*The tubes*, both in the cortex and pyramids, undergo marked changes. Those included in the masses of connective tissue are diminished in size, their epithelium is flattened, some contain cast matter, many are obliterated. The tubes between the masses of new connective tissue are more or less dilated; their epithelium is flattened, cuboidal, swollen, degenerated, or fatty. The dilatation of the tubes may reach such a point as to form cysts of some size, which contain fluid or coagulated matter. These cysts follow the lines of systems of tubes, or are situated near the capsules.

Of the *glomeruli* a certain number remain of normal size, but with the tuft cells swollen or multiplied. Many others are found in all stages of atrophy and of change into connective tissue. The atrophy seems to depend partly on the growth of tuft cells and intra-capillary cells, partly on the thickening of the capsules, partly on the occlusion of the arteries. If the chronic nephritis follows chronic congestion of the kidneys the glomeruli remain large, with an increased growth of tuft cells, or they become atrophied, but with the dilatation of the capillaries still evident. The capillaries of the glomeruli may be the seat of waxy degeneration. The arteries exhibit the same changes as have already been described in speaking of chronic exudative nephritis.

## COMPLICATING LESIONS.

*Heart.*—Hypertrophy of the left ventricle of the heart is frequently caused by exudative nephritis, but much more frequently by chronic nephritis without exudation. It must be admitted, however, that such an hypertrophy, although frequent, is not constant, that with both exudative and non-exudative nephritis there may be no change in the wall of the left ventricle. The hypertrophy of the wall of the ventricle may after a time be succeeded by dilatation, or chronic degeneration, or myocarditis.

Chronic endocarditis is often associated with this form of nephritis, apparently both lesions being produced by the same causes. It may also happen that chronic endocarditis causes first chronic congestion of the kidneys and then chronic nephritis without exudation.

*Lungs.*—Pulmonary emphysema and cirrhosis of the liver are frequently associated with chronic nephritis.

*Arteries.*—One of the most important of the complicating lesions is chronic endarteritis. The relationship between endarteritis, or, more properly speaking, arteritis and nephritis, and the ways in which they are associated together, are not as fully understood as they should be. The principal reason for this is the failure to recognize the fact that chronic inflammation of the walls of the arteries is just as much a disease as chronic endocarditis, or emphysema, or cirrhosis of the liver.

*Arteritis.*

Unquestionably arteritis is more often seen associated with other diseases than by itself. It must also be admitted that it is of such frequent occurrence in old persons that it is natural to think of it as a senile change. Still farther, the use of such names as arterio-capillary fibrosis and arterio-sclerosis have helped to prevent us from classing arteritis with the other chronic productive inflammations.

Chronic inflammation may involve the entire aortic system of arteries, or it may be confined to a part of that system.

If only the arteries in some one part of the body are involved, then in that part of the body the blood-supply is irregular or cut off, the diseased artery may become dilated, or it may rupture.

If a large part of the aortic system of arteries is involved,



then the patients suffer from symptoms which seem to depend partly upon the changes in the arteries, partly upon attacks of contraction of the arteries, partly upon hypertrophy of the left ventricle of the heart and heart failure, partly upon the obstruction to the passage of blood through the cerebral arteries.

The clearest idea of general arteritis as a disease is to be obtained by observing it in persons not over forty years old who have no complicating lesions.

At first for a number of years these patients only suffer from impaired nutrition, a disposition to become anæmic, and attacks of dyspnœa. It can be seen and felt that the walls of the temporal and radial arteries are thickened and that the left ventricle of the heart is hypertrophied. At the times when the patient has dyspnœa the tension of the pulse is much increased.

For a considerable length of time the nutrition and the anæmia can be improved by climate and by diet. The attacks of dyspnœa can be controlled by the drugs which dilate the arteries. But sooner or later the patients get worse. Some of them get up a dyspnœa that cannot be controlled, the action of the hypertrophied heart fails, and the patients, after suffering for weeks or months with the most distressing symptoms, die. In other cases death takes place with cerebral symptoms—sudden unconsciousness, or aphasia, or hemiplegia. After the death of these patients no lesions of any consequence are found except the changes produced by chronic inflammation of the walls of the arteries.

It is evident that the symptoms and death of these patients are due to the changes in the arteries, that the disease from which they have suffered is chronic arteritis. But it is also evident that their symptoms—loss of nutrition, anæmia, contraction of the arteries, hypertrophy of the left ventricle, dyspnœa, heart failure, unconsciousness, aphasia, hemiplegia—are also the symptoms of chronic nephritis.

Still further we find that many patients with these symptoms do have both arteritis and nephritis. In any given case with these symptoms, therefore, it is a matter of importance to determine whether the patient has arteritis alone, or nephritis alone, or both diseases at the same time.

Patients who have chronic nephritis are more liable than are other persons to attacks of pericarditis, bronchitis, and gastric catarrh.

## SYMPTOMS.

*The Urine.*—The typical urine of chronic non-exudative nephritis is a urine increased in quantity, of a specific gravity of about 1.010, containing a diminished quantity of urea, without albumin or casts, or with a trace of albumin and a very few casts. But exacerbations of the nephritis and changes in the circulation may for a time considerably increase the quantity of albumin and the number of casts.

Very important modifications of the urine, however, are of ordinary occurrence. It is quite possible, with nephritis of this type far advanced, to have urine not below 1.023 in specific gravity and without albumin or casts. When one sees this urine during life and then the kidneys after death, it is difficult to understand how they can belong to each other.

On the other hand, there are cases in which the specific gravity of the urine falls almost to 1.000, either with or without waxy degeneration of the blood-vessels. In some cases the quantity of urine is very much increased—several quarts in the twenty-four hours. During the attacks of contraction of the arteries, to which these patients are liable, the urine may be diminished to a few ounces or even suppressed.

*Cerebral Symptoms.*—In a great many of the cases cerebral symptoms are developed at some time in the course of the disease. Headache and sleeplessness are often present, the headache sometimes so severe and continuous that the patient is nearly maniacal. Instead of the headache there may be neuralgic pains in different parts of the body.

Muscular twitchings and general convulsions are much more serious. They may be early symptoms, or not occur until late in the disease.

Hemiplegia, with or without aphasia, may be the first symptom to call attention to the nephritis, or may not occur until later in the disease. The invasion of the hemiplegia is sudden and is usually accompanied by coma. There is loss of motion alone, or of both motion and sensation. The hemiplegia, aphasia, and coma may continue up to the time of the patient's death, or disappear after a few hours or days. In the latter case the patient may have several such attacks. These attacks have been ascribed to localized œdema of the brain. In the cases which I have seen

there were no changes in the brain tissue, but the cerebral arteries were damaged by chronic arteritis.

Delirium, mild or violent, stupor, and coma may come on in sudden attacks, or be developed slowly and gradually.

When these cerebral symptoms come on in attacks the pulse is of high tension, the temperature is raised, and the patients are said to suffer from acute uræmia. Very often they recover from a number of these attacks. In the fatal attacks the pulse often loses its tension and becomes rapid and feeble; the patients die comatose with a feeble heart.

Instead of such acute attacks of cerebral symptoms, delirium and stupor may come on gradually in persons far advanced in their nephritis. The temperature is then apt to be below the normal and the pulse is rapid and feeble.

Temporary blindness, neuro-retinitis, or nephritic retinitis are developed in a moderate number of the patients.

Chronic bronchitis and emphysema very frequently exist and their symptoms often form a large part of the clinical history.

Attention has already been called to the large share that chronic arteritis may have in the production of some of the renal symptoms.

*The Heart.*—The left ventricle of the heart regularly becomes hypertrophied after the nephritis has lasted for several months. The disposition to hypertrophy is, I think, rendered greater by repeated attacks of contraction of the arteries and by complicating arteritis. The hypertrophy is usually easily made out. The patient remains unconscious of its existence, or has disturbances of sensation and palpitation. As the disease goes on the hypertrophied heart may become feeble, and then dyspnœa and the other evidences of feeble circulation make their appearance.

In the same way the complicating endocarditis, which so often exists, may give no trouble until the valves are a good deal changed, or the ventricles dilated, or the heart's action altered, or the arteries contracted; then the circulation is interfered with, and the results of venous congestion of different parts of the body show themselves.

*Dyspnœa* is a frequent symptom, often the first symptom noticed by the patient. It is a spasmodic dyspnœa coming on in attacks, which last for minutes, hours, or days. It is made worse by bodily or mental exertion, or by the recumbent position. It does not resemble bronchial asthma. It is apparently due to the

association of changes in the arteries and heart. It cannot be distinguished from the dyspnœa which is caused by arteritis without nephritis. With contraction of the arteries alone, or with a feeble heart alone, no dyspnœa may exist; but if the contraction of the arteries be so great that the hypertrophied heart cannot overcome the obstruction, or if with contraction of the arteries the heart becomes dilated or feeble, then the attacks of dyspnœa begin. At first the attacks are not severe and are of short duration, but if the mechanical conditions which cause them cannot be controlled, they become longer and more distressing.

*The stomach* may continue to perform its functions fairly well, but more often there is gastric indigestion, gastric catarrh, or spasmodic vomiting.

*Dropsy* as a rule is absent with non-exudative nephritis, unless it is complicated by chronic endocarditis, by cirrhosis of the liver, or by the disturbances of circulation which come on later in the nephritis.

Profuse bleeding from the pelvis of atrophied kidneys is sometimes seen. In all cases, after a time, the nephritis exerts its effects upon the nutrition of the patient, and the flesh and strength are diminished. On the other hand, the patients do not usually become as pale as they do with an exudative nephritis.

#### COURSE OF THE DISEASE.

It is characteristic of the chronic productive inflammations of the lungs, the heart, the arteries, the liver, and the kidneys that, while they often exist as serious and fatal diseases, they may also exist as lesions and yet do not interfere with long life and apparent good health. This seems to depend, at least in part, on the rapidity with which the inflammatory changes in these different parts of the body are developed. If the development is slow enough, the functions of the organ continue to be performed in spite of the new growth of connective tissue.

We have to admit that in all cases of chronic non-exudative nephritis a period of weeks, or months, or years elapses during which the changes in the kidney are slowly going on, and yet the patients seem well and are not aware that they have any disease. How far the nephritis can advance and how many years it can exist before the symptoms of it appear, it is difficult to say. We see a great many different stages in the development of the ne-

phritis in persons who die from other diseases and have never given any renal symptoms.

The nephritis is of slow development, gradually altering the structure of the kidney more and more, so that we should expect that the symptoms of the nephritis would also be developed gradually. This is very often the case, but quite as often the nephritis will advance without symptoms up to a certain point and then the patient suddenly becomes ill.

*A. Cases with Slow Development of Symptoms.*—Of the patients in whom the symptoms are gradually developed we may distinguish :

1. Patients who gradually develop hypertrophy of the left ventricle of the heart, with a lowering of the specific gravity of the urine, and a pulse that is easily made too tense ; otherwise their health is good. We often watch these persons for many years, expecting other renal symptoms. But the symptoms do not come, and the patients die of some other disease.

2. Patients who have digestive disturbances, and gradual loss of flesh and strength. The urine is of low specific gravity and increased quantity, or the specific gravity and quantity remain almost normal ; often from time to time there are traces of albumin and a few hyaline casts. These patients are often very puzzling. From year to year they slowly get more feeble and more emaciated ; the digestive disturbances are sometimes better and sometimes worse. Occasionally there is an interval of great improvement, so that the patients think they have entirely recovered. As the disease lasts a long time the patients are apt to see a number of physicians and get a number of opinions, for the diagnosis is really a difficult one. Some of the patients die from intercurrent diseases, but others go on and die simply exhausted with nothing but the chronic nephritis.

3. Patients who for months or years have attacks of spasmodic dyspnœa, and between these attacks are comparatively well. The patients are usually over forty years of age. The attacks of dyspnœa are apt to come on in the early morning and go off later in the day. Often chronic arteritis, or chronic endocarditis, exists at the same time. For a while the attacks of dyspnœa can be relieved, and the patients are capable of mental and physical exertion and feel quite confident of recovery. But as the attacks of dyspnœa recur they last longer and are harder to relieve. Finally comes the time when the dyspnœa cannot be

relieved. It lasts day and night the patients cannot lie down, the scrotum and legs become œdematous, and death hardly comes soon enough to relieve their distress.

4. Patients who have symptoms progressing for several years. At first vomiting, or headache, or neuralgic pains. Then dyspnœa, a little dropsy of the legs, and loss of flesh and strength. Finally death from exhaustion, or with an attack of convulsions, or in coma.

5. Patients in whom the symptoms come on in attacks, each attack worse than the preceding, and the general health more and more impaired between the attacks. During the attacks there are headache, sleeplessness, delirium, stupor, coma, convulsions, dyspnœa, vomiting—sometimes one, sometimes another the prominent symptom. The tension of the pulse is considerably increased. The urine is of low specific gravity and often contains a little albumin. Between the attacks the patients at first seem to be fairly well, but later they gradually lose flesh and strength. The urine between the attacks is of low specific gravity and contains little or no albumin. The patients finally die in one of the attacks.

*B. Cases with Rapid Development of Symptoms.*—Of the patients who are apparently in their ordinary health until there is a violent invasion of symptoms, we may distinguish those in whom the attack seems to be precipitated by an injury or an intercurrent disease, and those in whom it comes on without discoverable cause. In either case the attack regularly takes the form of cerebral symptoms, or of dyspnœa, or of vomiting, or of sudden death. During these attacks the tension of the pulse is high, the urine is diminished in quantity or suppressed and often contains a little albumin.

1. The cerebral symptoms are general convulsions, coma, hemiplegia, and aphasia. The convulsions come on suddenly; they are repeated several times; between them the patients are unconscious. Many of the patients die with the convulsions, but a few recover. We are apt to see these persons for the first time while the convulsions are going on, and are told by their friends that they were in their ordinary health until the convulsions began.

The coma is developed in the same rapid way. The patient is found in bed, in a room, or in the street, at first stupid and muttering incoherently, then completely comatose. From this

coma they do not emerge, but go on and die in a few hours or days.

The hemiplegia is like that with a clot or with an obstructed artery. The patient falls to the ground unconscious and hemiplegic. If the hemiplegia is on the right side there is usually aphasia. The paralyzed side of the body may remain quiet, or become rigid, or be moved involuntarily. The hemiplegia and unconsciousness usually continue up to death. But occasionally we see a patient who recovers both motion and consciousness.

2. The dyspnœa often starts with an ordinary bronchitis. The patients cannot lie down, they suffer from the constant feeling of dyspnœa, the pulse is full and tense, the drugs which usually dilate the arteries are of little or no effect, the scrotum and legs become œdematous. The patients only live a few weeks.

3. The vomiting may at first resemble that of acute gastritis, or that caused by some irritating substance in the stomach. But it continues, it is exhausting, it does not yield to the ordinary remedies directed to the stomach; the pulse is full and tense. Such vomiting, however, can often be stopped by the drugs which dilate the arteries.

4. The patients after an injury, or a surgical operation, or without discoverable cause become feeble, the heart's action is feeble, the urine is diminished or suppressed, and in a few hours the patient is dead. These cases are not common. They are very disagreeable for the physician, as the patients seem to die without sufficient cause.

#### TREATMENT.

The progress of the nephritis can be favorably affected by attention to the diet and mode of life, and to climate. As regards the diet, the quantity of sugars and starches taken should be restricted, and the ingestion of fats encouraged. The use of wine, spirits, and tobacco should be discontinued. Exercise in the open air is to be advised as long as the strength permits of it. As regards climate, we must consult the idiosyncrasy of the patient; it should be a climate where he eats well, sleeps well, and feels well. There is a decided advantage in not remaining in the same place throughout the year.

In the patients belonging to group one, with urine of low specific gravity and hypertrophy of the left ventricle, it will be

found that whenever the tension of the pulse is increased the patients do not feel quite so well. When this is the case potassium iodide will often soften the pulse and remove the discomforts. These patients can also be much improved by regulated exercise in the open air.

In the patients belonging to group two the treatment is directed to the digestive disturbances and the nutrition. The regulation of the diet and the mode of life, lavage of the stomach, relieving constipation, and increasing the production of bile are all of importance. When the production of urine is largely in excess of the normal, combinations of *nux vomica* and sodium bromide will sometimes act as a specific in reducing this undue quantity.

In the patients with attacks of spasmodic dyspnoea much can be done with the drugs which dilate the arteries and stimulate the heart. According to the tension of the pulse and the strength of the heart's action, we use these drugs separately or together. Chloral hydrate, nitro-glycerin, and potassium iodide are the most reliable of the arterial dilators; digitalis, strophanthus, and caffeine are the best cardiac stimulants for this purpose.

The treatment of the attacks of headache, convulsions, coma, hemiplegia, and vomiting is a matter of importance. The only working theory that one can go on is to believe that at the time of these attacks there is an irritant poison in the blood which causes contraction of the arteries, and that the cerebral symptoms are due partly to the contraction of the arteries and partly to the poison itself. What the poison is or whether it is in all cases the same poison we do not know.

Evidently the indications given for treatment by this theory are, first, to remove the poison from the blood and, second, to dilate the contracted arteries.

The plans which are ordinarily used to remove the poison from the blood are general blood-letting, purging, sweating, and diuresis. These measures unquestionably can do much good. Whether they do so because they remove poison from the blood, or because they relieve the arterial tension, is a matter open for discussion.

Dilatation of the arteries can be effected by hypodermic injections of morphine; by nitro-glycerin, chloral hydrate, and potassium iodide; and by sweating. Very often with these reme-



dies the pulse will become soft and the cerebral symptoms will disappear. But after the nephritis has advanced beyond a certain point it is found that all these remedies are inert; the tension of the pulse and the cerebral symptoms continue. Or, instead of this, the pulse loses its tension, becomes rapid and feeble, the cerebral symptoms continue, and the patients die.

#### PUERPERAL ECLAMPSIA.

During the later months of pregnancy, during labor, and immediately after childbirth, women not infrequently become anæmic and dropsical, have albumin in the urine, and develop alarming cerebral symptoms: headache, blindness, convulsions, and coma.

These symptoms are especially frequent in primiparæ, in young women, and with twin pregnancies. They may be repeated in several successive pregnancies. They belong to the second half of pregnancy, increase in severity as the pregnancy advances, and are at their worst during labor.

#### MORBID ANATOMY.

In women dying with cerebral symptoms and albuminuria at about the time of childbirth, I have found the kidneys in the following conditions:

Normal kidneys.

Dilatation of the pelves and ureters.

Acute degeneration of the kidney.

Acute exudative nephritis.

Acute productive nephritis.

Chronic nephritis.

In some of the patients, even in young women, I have found well-marked disease of the cerebral arteries.

#### ETIOLOGY.

A number of theories have been entertained as to the causes of puerperal eclampsia. It must be confessed that no one of them is satisfactory, and that the subject is still obscure. The ordinary explanations are as follows:

1. Pressure on the renal veins by the gravid uterus produces

a chronic congestion of the kidneys, which interferes with their functions.

2. Pressure of the uterus on the ureters renders it necessary for the kidneys to secrete against a higher pressure, so that they are unable to get rid of the proper quantity of excrementitious substances.

3. The kidneys are obliged to excrete waste products not only from the mother, but also from the enlarged uterus and the foetus, and this extra work they are unable to perform.

4. It has been demonstrated in some cases that before the convulsions there is a diminished quantity of excrementitious products in the urine, and after the cessation of the convulsions an increase of these products; therefore the convulsions are due to the retention of these excrementitious substances in the blood.

5. That for some reason the patients have cerebro-spinal congestion.

6. That for some reason they have cerebro-spinal anæmia.

7. That the convulsions are of the nature of acute epileptic attacks due to irritation of nerves in the pelvis.

8. That the enlarged uterus acts as an irritant to the vaso-motor nerves and so causes a contraction of the arteries throughout the body.

9. That the enlarged uterus causes irritation of the vaso-motor nerves which supply the renal arteries; the contraction of the renal arteries causes death or degeneration of the renal epithelium; the changes in the renal epithelium render the kidneys unable to excrete poisonous substances; the accumulation of these substances in the blood causes the convulsions, etc.

10. There is at the time of childbirth in some women a toxic substance produced in some unknown way, which is not caused by any change in the function of the kidneys, but which is capable of causing transudation of serum from the vessels, contraction of the arteries, acute degeneration of the kidneys, and acute nephritis. In other words, the changes in the kidneys are not the cause of the convulsions, etc., but they are the result of the same poison which produces the albuminuria and the cerebral symptoms.

#### SYMPTOMS.

(1) There are a considerable number of pregnant women in whose urine during the latter months of pregnancy albumin is

present in appreciable quantities; they have no other symptoms and pass through labor without trouble.

(2) There are women who, during the latter months of pregnancy, have scanty and albuminous urine, more or less dropsy of the legs, and become pale and anæmic. They may pass through childbirth safely and do well afterward, but some of them have a chronic nephritis dating from the pregnancy.

(3) In a small number of women at about the time of childbirth, either before, during, or after labor, there are cerebral symptoms. In some of these women albumin has been present in the urine during the pregnancy; in others, besides the albuminuria, dropsy and anæmia have also been present; but in others the cerebral symptoms are suddenly developed without any premonitory conditions.

The cerebral attacks are characterized by nausea and vomiting, headache, blindness, muscular twitchings, general convulsions, stupor, coma, hemiplegia, a rise of temperature, a pulse of high tension, venous congestion of the skin, the urine diminished in quantity or suppressed, usually containing large quantities of albumin. The cases vary as to how many of these symptoms are present. A fair proportion of the patients survive these attacks, although the children usually die. In the fatal cases death takes place with the general convulsions, with hemiplegia, or with coma.

After the termination of the labor and the disappearance of the alarming symptoms the anxieties of the obstetrician are at an end, but those of the physician begin. For in many of these women a nephritis originates during pregnancy, which continues afterward as a chronic inflammation and ultimately destroys life.

#### TREATMENT.

While there have been many different opinions as to the nature of puerperal eclampsia, there is a good deal of uniformity as to the treatment. If the convulsions come on at about the end of pregnancy, it is generally agreed that labor should be brought on and the child delivered as soon as possible. Apart from this we try to unload the veins and dilate the arteries. This can be done by general blood-letting and by the use of nitro-glycerin, chloral hydrate, and opium. In order to guard against the cerebral symptoms it is of more practical importance

to watch the arteries and the heart than to test the urine for albumin.

### SUPPURATIVE NEPHRITIS.

Suppurative inflammation of the pelvis of the kidney and of the kidney itself occurs under several different conditions. It is the result of injuries ; it is due to emboli ; it occurs without discoverable cause ; it is secondary to cystitis, the cystitis being due to stricture of the urethra, to stone in the bladder, to paraplegia, to operations on the urethra, bladder, and uterus, to gonorrhœa, or to enlarged prostate. Chronic suppurative pyelo-nephritis may be caused by the presence of calculi in the pelvis of the kidney.

#### I. SUPPURATIVE NEPHRITIS FROM INJURY.

Gunshot wounds, incised or punctured wounds, falls, blows, and kicks are the ordinary traumatic causes. If the injury be a very severe one, it causes the death of the patient in a short time ; if it be less severe, suppurative inflammation may be developed.

The inflammatory process may be diffuse, so that the whole of one or both kidneys is converted into a soft mass composed of pus, blood, and broken-down tissue ; or it is circumscribed, and one or more abscesses are found in the kidney, which may communicate with its pelvis.

*Symptoms.*—Rigors mark the beginning of the suppuration and are often repeated throughout its course. A febrile movement is developed, which is apt to assume the hectic character with sweating. There is often vomiting. There may be very severe pain referred to the region of the inflamed kidney. The urine is diminished or suppressed ; it contains blood alone, or blood and pus. In the bad cases the patients pass into the typhoid state, become delirious, and die comatose, or with a rapid and feeble pulse. Or the disease is protracted, the patients become more and more emaciated, and finally die exhausted. In other cases the symptoms abate, the urine returns to its natural condition, and the patients recover.

*Treatment.*—The management of these cases is rather surgical than medical. The external wound is to be treated antiseptically, and the suppurating kidney is to be incised or removed, as may be necessary.

## 2. ABSCESSSES PRODUCED BY EMBOLI.

In ordinary endocarditis with vegetations on the valves it often happens that fragments of the vegetations become fixed in the branches of the renal artery. When this is the case white infarctions are produced.

With malignant endocarditis and with septic infections emboli find their way into the branches of the renal artery and set up circumscribed foci of suppurative inflammation. The kidneys become enlarged and are studded with little white points surrounded by red zones. These little white points are formed by an infiltration of pus-cells between the tubes, followed by the death and breaking down of the kidney tissue. The bacteria of suppuration are found in these little abscesses.

*Symptoms.*—These embolic abscesses can hardly be said to have a clinical history. Whatever symptoms may belong to them are lost in those of the general disease from which the patient is suffering.

## 3. IDIOPATHIC ABSCESSSES.

These occur without discoverable cause. Only one kidney is involved. We find after death part of the kidney destroyed; the remaining portions contain abscesses; the pelvis is dilated and contains pus, the capsules are thickened, the suppurative inflammation may extend to the surrounding tissues so that sinuses are formed, and even perforations into the intestine or through the diaphragm. It is very difficult in these cases to tell whether the inflammation begins in the kidney or in its pelvis.

*Symptoms.*—The symptoms begin gradually and are for some time obscure. There are repeated chills and an irregular febrile movement. The patients lose flesh and strength, become anæmic, and are often troubled by nausea and vomiting. There is more or less pain over the inflamed kidney. After a time the pelvis of the kidney may be so much dilated as to form a tumor. If the pus escapes from time to time through the ureter, this tumor will vary in size. The urine at intervals contains pus and fragments of broken-down kidney tissue. If the suppuration extends, there will be sinuses running behind the peritoneum, or into the colon, or upward through the diaphragm. The disease is apt to last a long time. The patients are liable to have

chronic nephritis of the other kidney, or waxy degeneration of the viscera.

*Treatment.*—The only plan of treatment is to cut down on the suppurating kidney and treat it as an abscess, or to remove it altogether.

#### 4. SUPPURATIVE PYELO-NEPHRITIS WITH CYSTITIS.

Both kidneys become inflamed. The pelves are congested and coated with pus or fibrin. The kidneys are swollen, congested, and studded with foci of pus. The smallest foci are not visible to the naked eye, but with the microscope we find collection of pus-cells between the tubes, with swelling and degeneration of the epithelium within the tubes. The larger purulent foci look like white streaks or wedges running parallel to the tubes and surrounded by zones of congestion. The larger abscesses replace considerable portions of the kidneys.

The ureters in some cases are inflamed, their walls thickened, their inner surfaces coated with pus or fibrin. The bladder presents the lesions of acute or of chronic cystitis.

*Etiology.*—This form of nephritis seems to be always secondary to a cystitis, the infection extending from the bladder through the ureters to the kidneys. The cases of cystitis in which a suppurative nephritis is likely to be developed are those due to strictures of the urethra, stone in the bladder, operations on the urethra, bladder, and uterus, paraplegia, gonorrhœa, and enlarged prostate.

*Symptoms.*—When the nephritis occurs with cystitis due to stone in the bladder, strictures of the urethra, or operations on the genito-urinary tract, the symptoms are much the same. The patient has first the symptoms belonging to the cystitis, then he is attacked with chills and a rise of temperature. The chills are repeated, the temperature is irregular and accompanied by profuse sweating. There is a rapid change in the general condition of the patient, he becomes more prostrated and emaciated from day to day. The face is drawn and anxious, the tongue dry and brown, the pulse rapid and feeble; delirium is developed, and the patient finally dies in the septic condition. The urine is diminished in quantity or suppressed; it contains blood, pus, and mucus derived partly from the bladder, partly from the kidneys.

Cases of suppurative nephritis due to a gonorrhœal cystitis are not common, but several of them have been observed. Murchison describes two cases, in both of which the cerebral symptoms were very marked—delirium, convulsions, and coma. I have seen one such case. The patient was a prostitute who came into the hospital with a specific vaginitis. After a few days she developed the symptoms of an acute cystitis; then, after a few more days, she was attacked with chills and a rise of temperature, passed rapidly into the septic condition, and died. At the autopsy there were found acute cystitis, pyelitis, and numerous small abscesses in both kidneys.

When suppurative nephritis complicates the cystitis due to enlarged prostate, the symptoms are somewhat different. The patients are usually men over fifty years old. They have generally suffered from the symptoms of enlarged prostate, retention of urine either constant or intermittent, and more or less cystitis with pus and mucus in the urine. Sometimes, however, no such history is obtained; the patients assert that they have had no previous bladder trouble. The first symptom is a diminution in the quantity of urine, with the appearance of blood mixed with it, or the urine may be suppressed altogether. The blood may be present in considerable quantities, so that the patients seem to pass blood instead of urine. The patients rapidly become prostrated and very anxious. There are usually no chills, and there may be no rise in temperature. The prostration becomes more marked, the pulse is rapid and feeble, the skin is cold and bathed in perspiration, and the patients die in collapse at the end of a few days. Or, instead of such a history, the patients may behave as if they were the subjects of septic poisoning.

*Prognosis.*—Suppurative nephritis secondary to cystitis is a very fatal disease; so far as I know all the patients die.

*Treatment.*—The treatment for these cases is altogether a preventive one directed to the cystitis. When the nephritis is once established we have no further control over the case.

#### TUBERCULAR NEPHRITIS.

The different portions of the genito-urinary tract—the kidneys, ureters, bladder, seminal vesicles, prostate, testicle, uterus, Fallopian tubes, and ovaries—may become the seat of a localized tubercular inflammation.

Such an inflammation may involve one of these organs, or several of them. If several of them are involved, they are on the same side of the body, usually the left side. The inflammation is attended with the growth of tubercle bacilli and the formation of tubercle tissue. The tubercle tissue soon dies and undergoes cheesy degeneration.

In the kidneys the inflammation begins in the mucous membrane of the pelvis and calyces, and extends to the parenchyma, until a large part of the kidney is replaced by the degenerated new tissue. The cheesy masses may soften or become calcified, while the kidney tissue between them is converted into fibrous tissue more or less infiltrated with pus.

The other kidney, after a time, is apt to become the seat of the exudative form of chronic nephritis with waxy degeneration of the blood-vessels.

The tubercular nephritis may be complicated by tubercular inflammation of other parts of the genito-urinary tract on the same side of the body, by tubercular peritonitis, pulmonary tuberculosis, or general tuberculosis.

The disease is said to occur at all ages ; it is most frequent in middle-aged persons. It occurs twice as often in men as in women.

#### SYMPTOMS.

The urine usually, but not always, contains from time to time blood, pus, detritus, epithelium, shreds of tissue, and tubercle bacilli. When the other kidney has become the seat of chronic nephritis the specific gravity of the urine falls and albumin and casts are present.

Pain, either continuous or in paroxysms, and tenderness are often present in the inflamed kidney. There may be hectic fever with night sweats ; the patients gradually lose flesh and strength. The kidney may be enlarged so as to form a tumor which can be felt. After a time there are added the symptoms of tubercular inflammation of other parts of the genito-urinary tracts, of tubercular peritonitis, of pulmonary phthisis, of waxy degeneration of the viscera, or of chronic nephritis of the other kidney.

The disease lasts, as a rule, for several years. Most of the cases terminate fatally, but it is possible for the inflammation to stop and for the patient to recover.



## TREATMENT.

The proper treatment for tubercular nephritis is the removal of the diseased kidney. The practical difficulty is to make the diagnosis before other parts of the genito-urinary tract have become tubercular, or before the remaining kidney has become the seat of chronic nephritis.

We may hope that climate and feeding may have the same good effects on tubercular nephritis as they have on pulmonary tuberculosis.

## NEW GROWTHS OF THE KIDNEY.

The most important new growths of the kidney are those which belong to the classes of sarcoma and adenoma.

*The sarcomata* grow from the kidney itself, or from its pelvis. They are composed of connective tissue with an excess of cells, with which may be mixed mucous tissue or muscular tissue. These tumors often reach a large size and may grow for a number of years before they cause death. They form a hard abdominal tumor which at first retains the position and shape of the kidney, but may finally become so large as to occupy a considerable part of the abdominal cavity.

They are found as congenital tumors, are rather frequent in infants and children, and are occasionally met with in adults.

*The adenomata* grow in the cortex of the kidney in the form of nodular tumors. They may follow the papillary or the tubular type. In some cases the tumor or tumors never attain any considerable size, are not malignant, and give rise to no symptoms. In other cases the tumors become much larger, and may then behave like malignant growths. These large tumors are very vascular. The adenomata which run a malignant course, with the formation of metastatic tumors, are often called carcinomata.

## SYMPTOMS.

The sarcomata and adenomata, so far as their symptoms are concerned, may conveniently be described together. In both of them there are four principal symptoms: a tumor, pain, hæmaturia, and loss of nutrition.

The tumor is appreciable as soon as it has reached a sufficient

size. While it retains the natural position and outlines of the kidney the diagnosis is comparatively easy, but as the tumor becomes larger and adhesions are formed, it becomes more difficult to distinguish it from other abdominal tumors.

Hæmaturia is present at some time in the disease in about half the cases. The hemorrhages may be very large with rapid anæmia and exhaustion, or moderate in quantity, or so little as only to be appreciable with the microscope. They are apt to recur at intervals of days or weeks.

The pain is referred to the situation of the diseased kidney. It is by no means a constant symptom, but it may occur early and be throughout a prominent feature. They are apt to come on in attacks and to radiate downward along the course of the ureter.

Loss of appetite, nausea and vomiting are troublesome symptoms in some of the patients.

The loss of flesh surely comes sooner or later, but it is curious in some cases how long the general health may remain unaffected and how long life can be prolonged even with enormous tumors.

#### TREATMENT.

The same rule seems to hold good for these tumors in the kidney as for the same tumors in other parts of the body. If the kidney be removed while the growth is still small, the prognosis is fairly good. If it be not removed until the tumor is large or until metastatic tumors have been formed, the prognosis is bad.

#### THE CYSTIC KIDNEY.

Cysts are formed in the kidney both during intra-uterine and extra-uterine life.

The congenital cystic kidney is a very remarkable pathological condition. Either one or both kidneys are enormously enlarged and converted into a mass of cysts. The cysts are of all sizes, and are separated from each other by fibrous septa or compressed kidney tissue. They contain a clear, yellow, acid fluid, holding in solution the urinary salts; or the fluid is turbid and brown, and contains blood, uric-acid crystals, and cholesteroline. The cysts are lined with a single layer of flat epithelial cells. They seem to be formed by a dilatation of the tubules and of the capsules of the Malpighian bodies. As causes for such a dilatation are found obliteration of the tubes in the papillæ, or

stenosis of the pelvis, ureters, bladder, or urethra. Other congenital malformations are often associated with this one.

In adult life we find three varieties of cystic kidney :

1. In kidneys, which are otherwise normal, there are one or more cysts filled with clear or brown serum, or colloid matter. These cysts do not appear to interfere at all with the functions of the kidneys.

2. In chronic diffuse nephritis, especially in the atrophic form, groups of tubes are dilated. Apparently one or more of the larger tubes in the pyramids are obstructed, and this causes dilatation of a corresponding group of tubes. Such a dilatation may be moderate in size, or it may form cysts visible to the naked eye.

3. Both kidneys are very much enlarged and converted into a mass of cysts containing clear or colored serum, or colloid matter. The nature of these cysts is uncertain. It is possible that they are congenital. They are sometimes associated with similar cysts in the liver. Their clinical history resembles that of some of the cases of chronic diffuse nephritis without exudation. The patients go on for a long time without symptoms, except that the specific gravity of the urine gradually falls and a little albumin is occasionally present. Finally they have an attack of general convulsions or of coma, and die in a short time.

#### PERINEPHRITIS.

The loose connective tissue around the kidney may become the seat of suppurative inflammation, and in this way abscesses of considerable size may be formed.

*Etiology.*—Perinephritis is either secondary or primary. The secondary cases are due to extension of the inflammation from abscesses in the vicinity, such as are formed with caries of the spine, pelvic cellulitis, puerperal parametritis, perityphlitis, sup-puration of the kidney, and pyelonephritis. The primary cases occur after exposure to cold, after contusions over the lumbar region, great muscular exertion, and without discoverable cause. The lesion is said to complicate typhus and typhoid fever and small-pox. The disease occurs both in children and adults; most of the cases reported have been in persons between the ages of twenty and forty years.

*Morbid Anatomy.*—The connective tissue behind the kidney

seems to be the usual point of origin of the inflammatory process, and it is here that the pus first collects. After the abscess has reached a certain size the suppuration seems to have a natural tendency to spread, and the pus burrows backward through the muscles; downward along the iliac fossa, even as far as the perineum, scrotum, or vagina; forward into the peritoneal cavity, the colon, or the bladder; upward through the diaphragm. The kidney is compressed by the abscess or becomes involved in the suppurative process. The soft parts around the abscess become thickened and indurated.

*Symptoms.*—The disease begins, as a rule, with pains and tenderness referred to one lumbar region, between the lower border of the ribs and the crest of the ilium, sometimes to a point above or below this. At about the same time are developed repeated rigors, a febrile movement with evening exacerbations, sweating, loss of appetite, vomiting, and prostration. These are all the symptoms for from one to two weeks. Then the skin over the lumbar region on one side becomes red and œdematous, the corresponding thigh is kept flexed and rigid, for any movement of it gives pain. Then the lumbar region becomes more and more swollen until fluctuation can be made out, and finally the abscess breaks through the skin. If such cases are left to run their course the abscess may reach a very large size. If the pus does not extend backward, but in some other direction, the symptoms are more obscure, for the local symptoms of an abscess in the back are absent.

If the abscess ruptures into the peritoneal cavity, the symptoms of acute general peritonitis are suddenly developed. If it perforates into the colon or bladder, the pus is discharged with the fæces or the urine. If the perforation is through the diaphragm there will be empyema, or the lung becomes adherent and pus is coughed up from the bronchi. As soon as the abscess is opened and the pus escapes, the acute constitutional symptoms subside.

Trousseau believes that the inflammatory process sometimes stops short of the production of pus. In such cases, of course, there are no evidences of the formation of an abscess.

If the abscess ruptures spontaneously or is opened by the surgeon, the patient is likely to recover, but the suppurative process may continue and the patient die exhausted, usually with waxy viscera.

Perforation into the peritoneum, the pleura, or the lung causes death.

*Treatment.*—The main point in the treatment is to discover the abscess and to open it. The longer the suppuration goes on and the larger the abscess, so much the worse is the prognosis. It is proper to explore with the aspirator after the disease has lasted for a few days, even if no fluctuation can be made out.

#### HYDRONEPHROSIS.

*Definition.*—Dilatation of the pelvis and calyces of the kidney.

*Etiology.*—Dilatation of the pelvis and calyces of one or of both kidneys can be produced by any mechanical obstruction to the escape of urine.

Such an obstruction may begin during foetal life, so that when the child is born both ureters and the pelves of both kidneys are found much dilated. Such children die soon after birth. The hydronephrosis in these cases is due to some congenital malformation, but occasionally we see this condition in children in whom it is very difficult to find the seat of obstruction. It is supposed that in these cases there exists a membranous obstruction which is broken by the probe used to explore the urethra.

In adults the mechanical cause of the obstruction to the escape of urine may be situated in the ureter, bladder, or urethra, or in the abdominal cavity near the ureters. According to the position of the obstruction, either one or both kidneys are affected.

*Morbid Anatomy.*—The pelves and calyces of the affected kidney are more or less dilated; the mucous membrane is thin and shining, or thickened. The kidney tissue becomes more and more thinned as the dilatation goes on, and after a time in the thinned kidney a chronic nephritis is set up. Then there is a growth of new connective tissue in the stroma of the pyramids and cortex; in some of the glomeruli there is a growth of the cells covering the capillaries, while other glomeruli are atrophied; degeneration of the renal epithelium and thickening of the walls of the arteries are also present.

*Symptoms.*—The patients suffer for a considerable length of time from the inconveniences belonging to the retention of urine. If the retention be due to stricture of the urethra or to disease of the bladder, the entire history is straightforward. Ob-

struction of the ureters, however, may give no evidence of their existence, and we find after death strictures of one or both ureters, for which we are unable to account.

It is not uncommon to find after death well-marked hydronephrosis of one or both kidneys in patients who have had no renal symptoms at all.

In some cases, after a time, there is developed in the compressed kidneys a chronic productive nephritis with a little exudation. Then the specific gravity of the urine gradually falls, a small quantity of albumin and a few casts make their appearance. The quantity of the urine remains normal, or is increased, or diminished, or suppressed. The patients may at any time have contraction of the arteries with an elevated temperature and cerebral symptoms, or they may pass into the condition of chronic uræmia with a feeble pulse and low temperature.

In other cases the most prominent symptom is the presence of a tumor in the abdominal cavity. The disease is then usually confined to one kidney. The tumor lies in the region of the loin, extending upward, downward, and forward as it increases in size, and pushing the intestines forward and to one side. The surface of these tumors may become inflamed and adhesions formed to the surrounding tissues. As they become larger they give rise to much discomfort and pain. The tumors feel like cysts. From time to time they may diminish in size, with an increased flow of urine from the bladder; they are not movable. If the disease is caused by renal calculi, there may be attacks of renal colic with bloody urine. If the fluid from the tumor is drawn off it usually contains urinary salts, but sometimes it is only clear serum.

In still other cases of hydronephrosis a suppurative inflammation attacks the pelvis of the kidney, and the patient suffers from pyelitis.

*Treatment.*—The treatment of hydronephrosis is altogether surgical. If possible the cause of the retention of urine must be removed. If large cysts are formed, they must be opened or removed.

#### PYELITIS.

It has already been mentioned, in describing suppurative nephritis, that with acute or chronic cystitis there may be an extension of the inflammation from the bladder to the pelves of the

kidneys, with suppurative inflammation of the pelves and of the kidneys themselves. In this form of acute pyelitis the inflammation of the kidney is more important than that of the pelves.

Calculi in the pelvis of the kidney often set up a pyelitis, which will be described with the account of renal calculi.

An acute catarrhal pyelitis is said to be caused by the use of turpentine and of cantharides, also by typhoid fever and the exanthemata. It lasts but a short time and gives rise to no symptoms of importance.

The important form of pyelitis is the chronic inflammation which most frequently follows cystitis, sometimes succeeds hydronephrosis, and rarely seems to be a primary inflammation. While in most cases the pyelitis is clearly secondary to the cystitis, it may very well happen that the bladder will get well, while the pyelitis continues. In the pyelitis which follows pregnancy it is not always easy to say whether the inflammation is due to the temporary hydronephrosis or to the cystitis caused by infected catheters.

*Morbid Anatomy.*—The mucous membrane of the pelvis and calyces is thickened, its stroma more or less infiltrated with cells, the layer of epithelium thickened in some places, thinned in others, its surface coated with mucus or muco-pus. The pelvis and calyces are more or less dilated : they may contain uric acid or oxalate of lime calculi, which have caused the inflammation ; or phosphatic concretions which are the result of it. In the kidney itself there may be a growth of connective tissue in the stroma of the pyramids and cortex, with degeneration of the epithelium and atrophy of the glomeruli, or a suppurative inflammation which may destroy considerable portions of the kidney.

*Symptoms.*—In the majority of the patients the symptoms of the cystitis, of the enlarged prostate, or of the stricture of the urethra are prominent features of the case.

So far as the pyelitis is concerned, the patients have pain and tenderness referred to the position of the diseased kidney. The urine from time to time contains blood, mucus, and epithelial cells from the pelvis of the kidney ; later in the disease many pus-cells are mixed with the urine, and sometimes phosphatic concretions. The urine is more frequently acid than is the case with cystitis. The pus may be discharged continuously, or the ureter may from time to time become occluded, and the urine

coming from the other kidney will be clear until the obstruction is overcome; then a large quantity of purulent urine will again be discharged.

In some patients the purulent inflammation of the pelvis continues; more or less suppurative inflammation of the kidney is added; there are also progressive loss of flesh and strength, chills, hectic fever, waxy degeneration of the viscera, and the patients finally die exhausted by the disease.

The distention of the pelvis of the kidney may be so great as to form a fluctuating, painful tumor of considerable size. If the ureter is not entirely occluded, the pus will escape from time to time with a corresponding change in the size of the tumor.

Occasionally, after a time the purulent discharge ceases, the pelvis contracts, the kidney is atrophied, and the patient gets well with one useful kidney. In some of the cases the secondary nephritis after a time gives symptoms. The specific gravity of the urine falls, the patients lose flesh and strength, become anæmic, have more or less dropsy, and finally have acute cerebral symptoms or pass into the condition of chronic uræmia.

If phosphatic concretions are formed in the pelvis, fragments are liable to come away from time to time and cause attacks of renal colic. Or it is possible for such fragments to become impacted in the ureters and cause fatal suppression of urine.

#### HEMORRHAGIC PYELITIS.

Cases characterized by intermittent attacks of pain over one kidney and bloody urine, or of bloody urine alone, are of not uncommon occurrence. Only a moderate number of these cases have found their way into print, but conversation with physicians and surgeons shows the existence of many unreported cases. The symptoms may follow either a mild or a severe course.

The milder form of the disease seems to be of most common occurrence in young girls. The only marked symptom is the appearance in the urine, for days or weeks, of red and white blood-cells and of epithelium from the pelvis of the kidney. The patients may also suffer from hysteria and from disturbances of digestion. After one or more of such attacks the patients recover altogether. No treatment is necessary except to relieve disturbances of digestion and improve the general health.



The severe form of the disease is seen both in males and females. From time to time the patients suffer from attacks of pain referred to one kidney, and at the same time the urine contains large quantities of blood. With the cystoscope the blood can be seen to come from the ureter belonging to the affected kidney. Some patients after a number of such attacks have no further trouble and recover entirely. In others, however, the pain is so intense, or the loss of blood so threatening, that surgical operations have been undertaken for their relief, usually with the expectation of finding calculi in the kidney. These kidneys have been simply cut down on and felt of, or needled, or split open, or extirpated. It is a curious fact that recoveries are reported after each one of these operative procedures.

In the only one of these kidneys which I have had the opportunity of examining after extirpation, the kidney tissue itself was normal and contained no blood in the tubes or stroma. The mucous membrane of the pelvis and calyces was much thickened, its stroma infiltrated with cells, and the epithelium irregular.

*Treatment.*—The stricture of the urethra, enlarged prostate, cystitis, or other cause of the pyelitis is to be removed as entirely as possible. For the chronic pyelitis the improvement of the general health, an out-of-door life in a suitable climate, and the use of such waters as those of Ems and Poland Springs are often of great service. A considerable number of drugs have been employed for the cure of pyelitis—the mineral acids, the tincture of the chloride of iron, the alkalies, the vegetable astringents, salol, belladonna, and others. In pyelitis in the female good results are reported from washing out the pelvis through the ureter. If the distention of the pelvis with purulent fluid is very great, or if the kidney itself undergo suppuration, the kidney must either be opened or removed. The cystoscope is of much service in locating the kidney in which the pyelitis is situated.

#### RENAL CALCULI.

The solid constituents of the urine may be precipitated in the pelves of the kidneys in the form of sand, gravel, or calculi. The calculi are formed of uric acid, of uric acid with a shell of oxalate of lime, of oxalate of lime, or of the phosphates. Calculi composed of cystin are of a light-yellow color and lustrous, looking something like beeswax; they are of rare occurrence.

Calculi composed of xanthin, of fatty or saponaceous matters, of carbonate of lime and of fibrin have been described in rare cases.

*Etiology.*—Renal calculi are found in persons of all ages; they are of more frequent occurrence in males than in females. A sedentary life, gout, any conditions which produce an exaggerated excretion of uric acid, of oxalate of lime, of phosphates, or of cystin, or inflammation of the pelves of the kidneys, may act as causes.

*Symptoms.*—Small calculi may be formed in the pelves of the kidneys, be passed through the ureters into the bladder, and finally escape through the urethra without giving any trouble.

Larger calculi, which are formed in the pelves and pass through the ureters, cause by their passage attacks of renal colic. During an attack of renal colic the patients have severe pain, coming on suddenly or gradually, referred to the position of the kidney and ureter and radiating down into the groin, or simply a diffuse abdominal pain. The testicle is retracted, sometimes painful and swollen. Occasionally the pain is so severe that the patients faint or have general convulsions. Vomiting or retching is sometimes frequently repeated and very distressing. There may be a moderate rise of temperature.

The urine, during the attack, is passed frequently, in small quantities, often with a good deal of pain, and may contain blood. After the attack a considerable quantity of urine is passed, which for a time may be bloody.

Most attacks of renal colic last only for a few hours, but they may be prolonged, with intermissions, for a number of days. The same patient may have one attack or several attacks.

The calculus, instead of getting through the ureter into the bladder, may become impacted. If the patient has only one kidney, or if the kidneys of both ureters are obstructed, the urine is entirely suppressed, and the patients die after a number of days in the condition of chronic uræmia. If the ureter of only one kidney is obstructed, either hydronephrosis or pyonephrosis is apt to follow.

The calculi, after being formed in the pelvis of the kidney, may remain there. When this happens the patient may never have any symptoms, nor any considerable change in the kidney; but without any symptoms the kidney may become atrophied. More frequently the patients have repeated attacks of pain over

one kidney and bloody urine. After a number of attacks they may finally pass a calculus. If the attacks cease and no calculus is passed, we cannot be certain whether the patient has a renal calculus or has had a hemorrhagic pyelitis.

Very often the calculus acts as an irritant and sets up inflammatory changes in the pelvis or in the kidney. Or instead of this, a pyelitis is followed by the formation of phosphatic calculi.

If there is a chronic pyelo-nephritis, the patients have pain and tenderness over the diseased kidney, either continuously or in attacks. The urine at intervals contains pus and blood, sometimes fragments of the calculi. The dilatation of the pelvis of the kidney may form a tumor of appreciable size. The patients lose flesh and strength, they have an irregular fever, they are liable to have chronic nephritis of the other kidney, or waxy degeneration of the viscera.

In some cases the calculus causes but little change in the pelvis of the kidney, but the kidney itself becomes the seat of chronic diffuse nephritis with a little exudation. The urine then becomes of low specific gravity and contains a little albumin. The patients are liable to attacks of contraction of the arteries, with dyspnœa or cerebral symptoms, or they may become somewhat dropsical.

*Treatment.*—To prevent the formation of uric acid and oxalate of lime calculi we regulate the diet by excluding starch and sugar. We insist on sufficient exercise in the open air. The patients should spend several weeks every year at one of the alkaline springs, or should take from time to time one of the alkalies largely diluted with water. Piperazin is recommended in doses of from 5 to 10 grains dissolved in large quantities of water. Urotropin is also given in doses of 20 grains daily, dissolved in water and taken at one dose in the morning.

The treatment of the attacks of renal colic is directed to the relief of the pain and to hastening the passage of the calculus. The means usually employed for this purpose are hypodermic injections of morphine, inhalations of ether and chloroform, and the hot baths. The use of doses of 30 drops of tr. belladonnæ every two to three hours, until slight delirium is produced, has been employed to cut short the attacks and favor the passage of the calculus.

A calculus in the pelvis of the kidney, or one impacted in the ureter, can only be removed by a surgical operation.

## GLYCOSURIA.

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Sugar may be present in the urine as a temporary condition, or persist in moderate quantity for some time, although the patients do not have diabetes.

Certain poisons may cause temporary glycosuria: curare, carbonic-acid gas, nitrite of amyl, morphine, chloral hydrate, cyanide of potash, sulphuric acid, mercury, alcohol, and chloroform.

With some of the infectious diseases sugar may be found in the urine: cholera in the stage of reaction, anthrax, diphtheria, typhoid fever, scarlatina, remittent fever, and cerebro-spinal meningitis.

Gastric catarrh, functional disorders of the liver, gout, and imperfect digestion of sugars and starches may be accompanied by the presence of sugar in the urine.

Sugar may also be found in the urine after mental emotions, trigeminal neuralgia, concussion of the brain, cerebral apoplexy, with atrophied kidneys, and during pregnancy.

Such a temporary presence of sugar is usually not of importance, but it may be followed by diabetes.

## DIABETES MELLITUS.

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*Causes.*—This is a disease characterized by an increased production of urine, by the presence of sugar in the urine, and by changes in the general health of the patient.

In adults the disease is twice as common in men as in women. It is especially frequent in young and middle-aged adults, but it is not rare in children and old persons. It is more prevalent among the well-to-do than among the poor. It is found in nearly all parts of the world, but is more prevalent in some localities than in others. There seems to be an inherited predisposition to the disease.

*Lesions.*—The brain is seldom entirely normal in persons who die from diabetes, but the changes found in it seem to be the result rather than the cause of the disease. The brain itself may be œdematous and congested; or softened; or studded with minute hemorrhages; or with enlargement of the peri-vascular spaces; or with small cysts in the white matter. The pia may be thickened.

In rare cases tumors of the medulla, the pons, or the cerebellum have been found, which have been looked on as the cause of the diabetes.

The heart muscle may be the seat of fatty degeneration or of glycogenic degeneration.

In the blood hæmoglobin and the red blood-cells are decreased, the sugar is increased in quantity.

The lungs may be simply congested and œdematous; more frequently they are the seat of a tubercular pneumonia; occasionally portions of them become gangrenous.

The liver may be infiltrated with fat, or cirrhotic. In a few cases abscesses have been found in it.

The pancreas is the only part of the body of which the diseases really seem to cause diabetes. Extirpation of the pancreas in

animals and in man seems to be regularly followed by a true diabetes. In persons who die from diabetes the pancreas is often found to be much changed by interstitial inflammation, with a growth of new connective tissue and degeneration of the epithelial cells.

The kidneys are usually more or less changed. They may be the seat of chronic degeneration, or of chronic nephritis with exudation. The epithelial cells of Henle's loops are often changed by hyaline degeneration, or contain masses of glycogen.

The nature of the disease is still involved in obscurity. Sugar is introduced into the body as part of the food, and is also formed in the tissues. Normally, this sugar is afterward changed into other substances. In diabetes, either there is a failure in this destruction of sugar, or too much sugar is produced in the tissues, or both these conditions are present at the same time.

*Symptoms.*—It has long been observed that there are mild and severe cases of diabetes, and that the differences between these two sets of cases are so striking that it is difficult to believe the patients are suffering from the same disease. On the other hand, mild cases may gradually change into severe ones, and there also exists an intermediate set of cases, neither very mild nor very severe.

In the mild cases the urine is considerably increased in quantity and contains a moderate quantity of sugar. The general health of the patients is but little affected. They complain of thirst, of disturbances of digestion, of a little loss of flesh and strength. If the sugars and starches are excluded from their food, the quantity of urine diminishes and the sugar disappears. If a strict diet is kept up for some time the patients may later be able to return to the use of sugar and starch, and still remain well.

In the intermediate cases the quantity of urine and of sugar is larger, and the loss of nutrition is much more marked. It requires the strictest diet, and sometimes in addition medical treatment, to entirely get rid of the sugar in the urine. It is much more difficult to get the patients into such a condition that they can return to an ordinary diet without a recurrence of the sugar in the urine.

In the severe cases the quantity of urine and of sugar is very large. The patients have a troublesome thirst and an unnatural

hunger. The gums become unhealthy and bleed easily, and the teeth decay. The mouth is always dry, and often coated with a tenacious mucus. The functions of the stomach, liver, and intestines are disturbed in various ways. The patients lose muscular strength, and gradually become more and more emaciated. Neither the strictest diet nor medical treatment will cause the sugar to disappear from the urine. After a time one of the complicating lesions will be developed—the furuncles, the tubercular pneumonia, the degeneration of the kidney, the chronic nephritis, the gangrene of the feet.

As a rule, these severe cases of diabetes begin slowly and gradually, and life is not destroyed until after a number of years. But occasionally we see cases in which the disease seems to run its course within a few months or weeks.

The patients who have the severe form of diabetes are liable at any time to have an attack of diabetic coma. The group of symptoms called by this name may follow one of three types :

1. After some unusual bodily or mental exertion the patients suddenly feel very weak. The skin is cold, the pulse is feeble, they become stupid, then comatose, and die in a few hours.

2. For from six to nine days the patients complain of weakness, constipation, loss of appetite, slight drowsiness, breathlessness, and pain in the abdomen. Then come on headache, restlessness, delirium, abdominal pain, rapid and labored breathing, cyanosis, a feeble and rapid heart, stupor, and coma. The patients die within a few days.

3. There is no dyspnœa or prostration, but sudden headache, vertigo, stupor, coma, and death within a few hours.

We are still ignorant of the exact nature of these very fatal attacks. It has been supposed that they are caused by changes in the nerve-centres, by the inspissation of the blood, by fat embolism, by uræmia, or by acetonæmia.

In the mild cases the prognosis is very good, in the intermediate cases it is doubtful, in the severe cases it is bad. Children do much worse than adults. Old people often bear the disease very well.

*Treatment.*—We usually try, first, the regulation of the diet and of the mode of life. If these are not successful, we must add the use of drugs.

The regulation of the diet consists in cutting off from the food the sugars and starches.

The patients may eat : Meat, fish, soups, almond, bran, or gluten bread, eggs, cream, butter, cheese, oil, spinach, turnips, beans, cauliflower, cabbage, asparagus, lettuce, celery.

They must not eat : Sugar, wheaten bread, rice, sago, arrow-root, tapioca, macaroni, potatoes, carrots, parsnips, beets, peas, onions, and fruits.

They should not drink wines or liquors.

It is necessary, however, to take the greatest care not to exclude the starches and fruits so rigidly that the patients are insufficiently fed.

The regulation of the mode of life consists in seeing that the patients lead an out-door life, with sufficient exercise.

The drugs which are ordinarily used are : The preparations of opium. The bicarbonate and salicylate of soda, about one hundred grains daily. The alkaline waters of Carlsbad and Vichy. The sulphide of calcium, one-quarter of a grain to two grains four times a day. Clemen's solution of bromide of arsenic, five to twenty minims three times a day. The carbonate of lithia and arseniate of soda dissolved in large quantities of water. Iodoform, one-half a grain to two grains three times a day. Jambol in powder, ten grains three times a day. Antipyrin, ten to twenty grains three times a day. Pilocarpine, one-twelfth of a grain twice a day.



## DIABETES INSIPIDUS.

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This is a disease characterized by the passage of large quantities of urine of low specific gravity, the condition not being due to disease of the kidneys.

*Causes.*—The disease usually occurs in young adults. It is more common in males than in females. We know nothing concerning its nature and causes.

*Symptoms.*—The patients pass large quantities of urine of low specific gravity. They complain of continual thirst, of loss of flesh, of disturbances of digestion, and of a variety of hysterical and nervous symptoms.

In some of the patients the disease is readily relieved by treatment ; in others it is persistent and annoying ; but even in the worst cases it is not fatal.

*Treatment.*—The drugs ordinarily used are ergot, gallic acid, the preparations of valerian, the mineral acids, and combinations of sodium bromide with nux vomica. Some patients are benefited by the exclusive diet of meat and hot water.

## HÆMOGLOBINURIA.

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### I. IDIOPATHIC CASES.

*Causes.*—This condition has been observed in persons of all ages, but more frequently in females than in males. Similar conditions can be produced experimentally in animals by injecting into their veins distilled water, glycerine, or pyrogallic acid. In man the attacks are often produced by a sudden cooling of the skin.

There is apparently, at the time of an attack, first a sudden destruction of red blood-cells and liberation of their hæmoglobin in the general circulation; second, an excretion by the kidneys of the hæmoglobin which has thus been liberated.

*The symptoms* manifest themselves in paroxysms. Between the paroxysms the urine is normal, and the patients seem to be well.

The paroxysms commence with chilliness, or a chill lasting from half an hour to two hours. At the same time nausea, vomiting, and more or less prostration are present. After the chill there may, or may not, be a rise of temperature. The first urine passed after the chill is of red or brown color, as if mixed with blood. It contains, however, no blood-cells, only hæmoglobin. The specific gravity of the urine remains unchanged; albumin and casts are present. The hæmoglobin is found in the urine for one or more days after the chill.

The attacks may be repeated several times a day, or at longer intervals. They may be repeated a number of times during a month, stop for several months, and then begin again.

The patients may also be troubled by disturbances of digestion and some loss of flesh and strength.

All of the patients seem, after a time, to recover.

*Treatment* is directed to improvement of the general health and avoidance of cooling the skin.

## 2. MALARIAL CASES.

A patient may have a number of ordinary paroxysms of intermittent fever, and then one or more paroxysms followed by the appearance of hæmoglobin in the urine.

The malignant forms of remittent fever are sometimes accompanied by the presence of hæmoglobin in the urine.

## PERITONITIS.

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### ACUTE PERITONITIS.

*Lesions.*—There are two anatomical forms of acute peritonitis: a productive inflammation with the growth of cells, and an exudative inflammation with the production of serum, fibrin, and pus.

1. CELLULAR PERITONITIS.—The peritoneum is congested and without its natural glistening appearance. There is an increase in the size and number of the endothelial cells, but neither fibrin, serum, or pus is present. The intestines are distended with gas.

2. EXUDATIVE PERITONITIS.—The peritoneum is at first congested. Its surface is coated with fibrin, or with fibrin and pus, which makes adhesions between the opposed surfaces of peritoneum. In the peritoneal cavity, or shut in by adhesions, are collections of purulent or brownish serum. The subperitoneal tissue may be infiltrated with pus-cells. The intestines are distended with gas. In the more prolonged cases there is a growth of new connective tissue-cells and a formation of permanent thickenings and adhesions after the absorption of the serum, fibrin, and pus.

*Causes.*—Cellular peritonitis seems to be caused simply by some irritant. Exudative peritonitis is caused by an irritant and by the growth of pathogenic bacteria.

The ordinary causes of peritonitis are:

Wounds and contusions of the abdomen.

Wounds, ulcers, new-growths, incarcerations, intussusceptions, ruptures, perforations, and inflammations of the stomach and intestines.

Inflammations of the vermiform appendix.

Injuries and inflammations of the uterus, ovaries, and Fallopian tubes.

Rupture and inflammation of the bladder. Inflammation of the kidneys.

Abscesses and hydatid cysts of the liver. Inflammation of the gall-bladder.

Inflammation of the spleen, pancreas, lymphatic glands, retro-peritoneal connective tissue, vertebræ, ribs, pelvic bones, and pleura.

Thrombosis of the portal vein. Peritonitis may complicate septicæmia, the infectious diseases, and Bright's disease. Occasionally we meet with cases of idiopathic peritonitis.

*Symptoms.*—The symptoms of the invasion vary with the character of the cause of the peritonitis. With an idiopathic peritonitis the invasion is sudden or more gradual.

More or less chilliness exists during the first day, and may be repeated later. The temperature rises, but runs a very irregular course. In some patients it is high throughout the disease; in others it rises and falls irregularly; in still others it will hardly be above 99° F.

The intestines become distended with gas, and the abdomen is swollen and tense. Such a tympanitic condition belongs regularly to general peritonitis, but it is also found with the localized forms of the inflammation.

In most cases pain and tenderness exist over those portions of the abdomen where the peritonitis is going on. The pain is increased by pressure, by motion, and by the act of breathing. The patients lie on their backs, with their knees drawn up, and move the diaphragm as little as they can. It is, however, not a very rare exception for the pain to be absent altogether.

There is vomiting of food, of green or brown fluid, or of stercoraceous matter. Often there is no active vomiting, but every few hours there is a regurgitation of brownish fluid and of whatever food the patient has taken. There is often much difficulty in getting the patients to retain any nourishment at all.

The bowels are usually constipated, but sometimes there is diarrhœa.

The pulse is rapid and feeble.

The breathing is rapid and shallow.

The patients rapidly lose flesh and strength.

The tongue becomes dry and brown.

The mind may remain clear, or alternating stupor and delirium are developed.

After childbirth there may be an infection of and through the uterus, which gives symptoms like those of peritonitis, although a real peritonitis is not always present.

1. A portion of the membranes or a blood-clot, by the third or fourth day after childbirth, may become putrid, with the growth of bacteria and the formation of ptomaines. The patients at once have chills, fever, marked prostration, pain and tenderness, and tympanites. If the putrid substance is expelled from the uterus, all these symptoms cease. If it is retained in the uterus a true peritonitis may be developed.

2. There is an endometritis with pus and fibrin coating the inner surface of the uterus, and infiltrating its wall.

3. There is thrombosis of the uterine veins. With either of these conditions there may be a pelvic, or a general, peritonitis. But even if the peritonitis is not present the symptoms are much the same.

*Treatment.*—We may treat an acute peritonitis as we would any other acute exudative inflammation, by rest in bed, the local application of cold, repeated small doses of calomel or the sulphate of magnesia, and small doses of opium.

Or we may adopt the opium treatment, which consists in giving such doses of opium, at regular intervals, as will keep the patient in a condition of semi-narcotism until the peritonitis has run its course.

Or we may employ surgical treatment, open the abdomen freely, and wash out the pus and fibrin.

**INFLAMMATION OF THE VERMIFORM APPENDIX.**—The vermiform appendix is given off from the inner and posterior aspect of the lower end of the caput coli. It is from three to six inches in length. It is composed of a peritoneal, muscular, and glandular coat. It may be turned upward behind the cæcum, or it may hang downward free in the peritoneal cavity.

*Lesions.*—1. There may be a catarrhal inflammation of the mucous membrane of the appendix; this usually gives symptoms of no great severity.

2. There is an exudative inflammation of the entire wall of the appendix. The wall is infiltrated with fibrin and pus, but there is no necrosis of the wall nor perforation. If the appendix is behind the cæcum, or if adhesions are formed early, the inflammation is localized. If the appendix is free in the peritoneal cavity and no adhesions are formed, a general peritonitis is soon established.

3. There is an exudative inflammation of the appendix, with necrosis, sloughing, and perforation at one or more parts of its wall. This is regularly attended with the formation of an abscess behind the cæcum, or in the peritoneal cavity.

4. The entire appendix becomes rapidly gangrenous with the formation of an abscess or a general peritonitis.

5. A patient may recover from the exudative inflammation, or from the inflammation with perforation, and then have subsequent attacks of the same character.

*Causes.*—The disease is most common in persons between the ages of ten and thirty years, but older persons are not exempt. In many cases a fecal concretion, or a foreign body, is found in the appendix, which may be the cause of the inflammation.

In typhoid fever, and in dysentery, there may be an inflammation of the appendix as a part of the lesions of either disease.

*Symptoms.*—These depend upon :

The inflammation of the appendix.

The localized peritonitis.

The formation of an abscess.

The general peritonitis.

The general infection produced by the infectious inflammation.

The cases group themselves as follows :

1. Cases which terminate in resolution. The patients may, for hours or days before the attack, complain of colicky pains, constipation, diarrhœa, or a diffuse pain over the entire abdomen. Or, without any such premonitory symptoms, the invasion is abrupt.

There are chills, or chilliness, a rise of temperature, vomiting, pain and tenderness in the right iliac region, within forty-eight hours a decided feeling of fulness and resistance in the right iliac region. These symptoms continue for one or two weeks and then subside, and the patients recover. The patients may have no further trouble ; they may have pain from time to time from the adhesions ; or they may have fresh attacks of inflammation of the appendix.

Such a course of the disease means that the inflamed appendix is situated behind the cæcum, and that no abscess is formed.

*Treatment.*—These patients do so well that operative treatment seems hardly necessary. Rest in bed, a fluid diet, emptying of the rectum by enemata, and the application of continuous cold over the right iliac region are the essential parts of the treatment.

2. Cases with the formation of an abscess. The symptoms begin, as in the preceding set of cases, with premonitory symptoms, or abruptly. But although the symptoms are the same, they are much more severe. By the end of a week the abscess is formed. There is now a more distinct tumor, and the patients give evidences of septic poisoning. The position of the abscess varies with that of the appendix. At any time a general peritonitis may be set up by extension of the inflammation or by rupture of the abscess.

The abscess may perforate into the peritoneal cavity, the bladder, or the colon. There may be suppurating tracts which extend backward, upward, or downward.

The patients die with evidences of septic infection or of general peritonitis. A small number recover with escape of pus into the colon, or its gradual absorption.

*The treatment* is to cut down on the inflamed appendix and remove it as soon as the diagnosis can be made. For in all these cases there is inflammation and perforation of the appendix and the formation of an abscess.

3. Cases in which there is an exudative inflammation of the appendix with or without perforation; the appendix is not behind the cæcum; no adhesions are formed; a general peritonitis is soon developed.

For a few hours, or for two or three days, the patients complain of malaise, nausea, vomiting, constipation or diarrhœa, and abdominal discomfort. They do not seem to be very ill, and are not confined to bed. Then comes suddenly the development of an acute general peritonitis, with marked prostration, fever, diffuse pain and tenderness over the abdomen, vomiting, tympanites, a brown, dry tongue, and an anxious face. The patients get worse rapidly and die in a few days.

*Treatment.*—The proper treatment is to operate and remove the inflamed appendix as soon as possible. The difficulty is to make the diagnosis before the development of the peritonitis. After the peritonitis is established, the operation is not often successful.

4. Cases in which the entire appendix becomes gangrenous within a few hours. The patients complain first of general malaise, chilliness, nausea, vomiting, constipation or diarrhœa, pain and tenderness in the right iliac region, and a rise of temperature. Within twenty-four hours the patients look as if they



were suffering from an infectious disease. The pain and tenderness in the right iliac region continue; there is a feeling of resistance in this region, and if an abscess is formed, a more distinct tumor. The temperature does not run very high, but the pulse is rapid and feeble, and the patients look more and more as if they were poisoned. The abdomen becomes tympanitic, the stomach ceases to retain food, and the patients die at the end of a few days.

*Treatment.*—An operation at the earliest possible moment is indicated in these patients. But even with an operation they are very unfavorable cases.

It is to be noted that some of the patients do not refer their pain to the appendix, but to some other part of the abdomen; that the appendicitis may complicate an acute colitis; that the secondary abscesses about the liver and the right lung may give more marked symptoms than the appendicitis; and that a secondary portal phlebitis may seem to be the primary lesion.

#### CHRONIC PERITONITIS WITH ADHESIONS.

This is a chronic productive inflammation without exudation, which involves more or less of the peritoneum, and results in the formation of new connective tissue in the shape of adhesions.

*Causes.*—Such a chronic peritonitis may follow an acute peritonitis; it may be due to abscesses in the abdominal cavity or in the wall of the abdomen; it may be a primary chronic inflammation without discoverable cause.

*Lesions.*—We find after death threads and membranes of connective tissue which join together adjacent surfaces of peritoneum in a great variety of ways. The cases vary as to the number and extent of the adhesions.

*Symptoms.*—With but few adhesions there are no symptoms, or only occasional attacks of pain, apparently brought on by flatulence.

With extensive adhesions, chronic catarrhal inflammation of the stomach and bowels, with their characteristic symptoms, are established. Often we can appreciate by palpation that the intestines are matted together. The patients lose flesh and strength, but seem to die rather from some intercurrent disease than from the peritonitis. In such patients there is always danger of strangulation of the intestine.

*The treatment* is directed principally to the disturbance of the functions of the stomach and bowels.

#### CHRONIC PERITONITIS WITH THE PRODUCTION OF SERUM, FIBRIN, PUS, AND CONNECTIVE TISSUE.

This is a chronic productive inflammation with exudation, which involves a considerable part of the peritoneum, and results in the formation of serum, fibrin, and pus, and of new connective tissue in the form of adhesions.

*Causes.*—It follows acute peritonitis; it is caused by diseases of the uterus and its appendages, by localized peritonitis, and by abscesses in the peritoneal cavity. It may begin as a chronic inflammation without discoverable cause.

*Lesions.*—The adhesions mat together the coils of intestine and divide up the abdominal cavity in a variety of irregular ways. The free surface of the peritoneum is coated with fibrin and pus. Purulent serum, in small or large quantities, occupies the peritoneal cavity or is shut in by adhesions.

*Symptoms.*—If the chronic peritonitis is secondary, there is the previous history of the localized peritonitis or abscess. If it is primary, the invasion is slow and obscure.

The patients suffer from more or less abdominal pain and tenderness.

The physical signs vary with the extent and distribution of the adhesions and the quantity of serum.

Constipation or diarrhœa are often present, but it is possible for the bowels to move regularly throughout the disease. In most patients there is an irregular febrile movement, but there may be no rise of temperature for months.

The heart's action and pulse are rapid and feeble.

There are symptoms of gastric dyspepsia, and the stomach becomes less and less able to digest and retain food.

The patients steadily lose flesh and strength, and die emaciated and feeble after a protracted illness.

#### CHRONIC PERITONITIS WITH THICKENING OF THE PERITONEUM.

This is a chronic productive inflammation with exudation, which involves the greater part of the peritoneum, and results in a diffuse thickening of the peritoneum and an accumulation of serum in its cavity.

*Causes.*—This form of peritonitis seems to be a primary inflammation, and to belong to adult life, but we are entirely ignorant as to its causes.

*Lesions.*—We find after death a general diffuse growth of dense connective tissue, forming a thickening of the parietal and visceral peritoneum. There may also be more or less serum in the peritoneal cavity.

*Symptoms.*—The only physical signs are those belonging to the presence of fluid in the abdominal cavity.

The symptoms depend largely upon the interference with the functions of the stomach, intestines, and liver.

The thickening of the peritoneal coat of the stomach is attended with contraction of the organ and catarrhal inflammation of its mucous membrane. The patients suffer from pain, vomiting, and failure of gastric digestion.

The intestines become the seat of a chronic catarrhal enteritis, with disturbance of the functions of the small and large intestine.

The thickening of the capsule of the liver diminishes the size of this organ, interferes with the portal circulation, and produces ascites.

The patients gradually lose flesh and strength, and die.

#### TUBERCULAR PERITONITIS.

The peritoneum is rather frequently the seat of localized tubercular inflammation. As is usually the case, the growth of tubercle bacilli and formation of tubercle tissue are associated with other forms of inflammation.

*Lesions.*—The tubercle tissue is in the form of miliary tubercles, of large cheesy masses, or of flat plates. The associated inflammation results in the production of serum, of fibrin, or of connective tissue in a variety of ways.

1. The peritoneum is thickly studded with miliary tubercles, coated with a little fibrin, and there is a little serum in the peritoneal cavity.

2. With miliary tubercles or cheesy nodules there is a very large exudation of serum, which distends the abdomen; or of fibrin, which fills in all the interstices between the abdominal viscera and also distends the abdomen.

3. With miliary tubercles, or with cheesy masses, there is a

considerable growth of new connective tissue. The new tissue may be confined to the omentum, and convert this into a hard tumor of some size.

Or it may simply form adhesions between the coils of intestine so that these become firmly matted together.

Or the adhesions may shut in collections of serum, or of pus, which may reach a considerable size and resemble cysts.

Tubercular pleurisy, and tubercular inflammation of the Fallopian tubes, are often associated with tubercular peritonitis.

*Causes.*—The disease occurs both in children and in adults. It is said to be most frequent in persons between twenty and forty years of age. It is most frequently a primary inflammation, but may be secondary to tuberculosis of other parts of the body.

*Symptoms.*—Few diseases present a greater variety in the development of the lesions and the manifestation of the symptoms than does tubercular peritonitis.

1. The latent form of the disease. There are cases in which not only are there no abdominal symptoms, but no apparent change in the general health. Operations for strangulated hernia and ovarian tumors from time to time disclose an extensive tubercular peritonitis of which no symptoms had been given. In the same way the disease may be discovered in autopsies on persons dying from other diseases.

There are cases in which there are no abdominal symptoms, but yet the general health suffers and death may follow. The appearance and feeling of the abdomen are natural. There is no evidence of fluid or of adhesions, no pain, no tenderness, no distention. The liver may be small and the spleen large. But there are a variety of rather indefinite symptoms. Loss of appetite, nausea, vomiting, constipation, diarrhœa, blood in the stools, a little jaundice, a rapid heart, an irregular fever with evening exacerbations, gradual loss of flesh and strength, sometimes a complicating meningitis, or pericarditis, or pleurisy. The course of the disease is slow, with long intervals of improvement. Recovery seems to be possible, but death is the rule.

2. The disease may behave at first like an acute peritonitis, with a sudden rise of temperature, vomiting, tympanites, and pain and tenderness over the abdomen. After some days these acute symptoms subside and the patients go on with a chronic peritonitis.

3. The patients behave as if they had a chronic peritonitis with a large exudation of serum. The abdomen becomes largely distended by turbid serum, and is the seat of more or less pain and discomfort. There is a slight and irregular rise of temperature. The patients vomit occasionally, the bowels are irregular, there is a gradual loss of flesh and strength.

4. In a tubercular peritonitis with an excessive production of fibrin the rational symptoms are the same as when there is an excessive production of serum. The abdomen is large, painful, and tender, but the physical signs are not those of fluid in the abdominal cavity.

5. The coils of intestine are matted together by connective-tissue adhesions, with but little or no fluid in the abdominal cavity. These are very chronic cases with little or no fever, and with symptoms largely referable to the stomach and intestines. They have loss of appetite, nausea, pain, vomiting, flatulence, diarrhœa, or constipation, and gradual loss of flesh and strength. The abdomen may be tympanitic or retracted; sometimes we can feel through the anterior abdominal wall the tumor-like mass made up of the adherent coils of intestine.

6. The tubercular inflammation involves chiefly the omentum. This gradually becomes thickened and rolled together until it forms a long, hard tumor running across the upper part of the abdomen. The clinical history of these patients is simply that they have a hard tumor in the upper part of the abdomen, with gradual loss of flesh and strength.

7. A sero-fibrinous or purulent exudation is sacculated by adhesions formed between the intestinal coils, the parietal peritoneum, the mesentery, and the abdominal or the pelvic organs. The rest of the peritoneum is altered by chronic inflammation. The collections of fluid occupy the entire anterior portion of the peritoneal cavity, or they are smaller and situated either in the middle of the abdomen or on either side. Such collections of fluid, when felt through the anterior abdominal wall, feel like tumors or cysts. They have frequently been mistaken for ovarian cysts.

These cases vary as to whether the symptoms of chronic peritonitis or those of an abdominal tumor predominate.

There may be an irregular fever, vomiting, constipation or diarrhœa, abdominal pain and tenderness, gradual emaciation, the patient always in bed, and getting steadily worse. Or the

constitutional symptoms are rather those of a malignant growth with a well-marked abdominal tumor. Or with a well-marked tumor the patients may continue to be well nourished.

The physical signs are those of one or more cystic or semi-solid tumors in the abdomen. The tumors are most frequently mistaken for ovarian cysts, or for malignant new-growths.

*Treatment.*—There seems to be good reason to believe that in some cases of tubercular peritonitis the inflammation runs its course, subsides, and the patients recover, but with permanent peritoneal adhesions.

In cases of tubercular peritonitis which have only lasted for a moderate length of time, with a large serous effusion and not a great many adhesions, a cure may be effected by making a free incision through the anterior abdominal wall, allowing the fluid to escape, and then closing the wound. In the older cases, with extensive and firm adhesions, operative interference is but of little value.

Apart from operation, the treatment consists in feeding the patients and relieving symptoms.

#### CARCINOMA OF THE PERITONEUM.

*Lesions.*—Primary carcinoma of the peritoneum occurs in the form of colloid cancer, or of hard nodules composed of a connective-tissue stroma enclosing cavities which contain cells of epithelial type.

The growths are regularly multiple, and grow in many different parts of the peritoneum. The tumors may all be of small size, or some of them reach a diameter of several inches; or, in the case of colloid cancer, very large tumors may be formed.

With the new-growth there is a production of serum, or of fibrin, or of connective-tissue adhesions, or a general thickening of the peritoneum.

*Causes.*—The disease belongs to persons over forty years of age.

*Symptoms.*—The physical signs depend upon the size of the tumors, the serum, and the adhesions.

The invasion of the symptoms is slow and gradual, and some time elapses before the patients seem to be seriously ill.

Disturbances of the stomach and intestines, more or less pain, and constant loss of flesh and strength are the prominent symptoms.

## RHEUMATISM.

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It is customary to speak of acute articular rheumatism, subacute rheumatism, chronic rheumatism, muscular rheumatism, gonorrhœal rheumatism, rheumatoid arthritis, scarlatinal rheumatism, and puerperal rheumatism as if they were all varieties of one and the same disease. We are, however, not sufficiently acquainted with the nature of most of these so-called rheumatisms to tell how nearly they are or are not related to each other.

### ACUTE ARTICULAR RHEUMATISM.

This is an acute, general disease of which the characteristic lesion is an inflammation of the joints and of some of the connective-tissue membranes.

*Lesions.*—The joint lesion is an acute exudative inflammation of the synovial membranes with congestion and an exudation of serum, but only rarely a production of pus or fibrin.

Of the connective-tissue membranes the pericardium and the endocardium are the ones most frequently inflamed ; less often the pia mater, the pleura, and the peritoneum.

There may also be inflammations of the iris, the pharynx and tonsils, and the lungs.

*Causes.*—The disease is most common in the temperate zones, but is also observed in both cold and warm climates. The attacks are not confined to any one season of the year.

The time of greatest liability to the disease is between the ages of ten and thirty years. There seems to be both an hereditary and a personal predisposition to the disease, and the same individual often suffers from several attacks. The strong and vigorous, and the feeble and debilitated, seem to be equally liable to the disease. Exposure to cold and wet may often act as exciting causes.

*Symptoms.*—The symptoms of the disease may begin in several different ways.

1. For a few hours, or for several days, the patients complain of general malaise, headache, irregular chills, a little fever, irritability, sleeplessness, a coated tongue, no appetite, urine loaded with urates, and irregular pains about the joints.

2. There are pain and stiffness of one or more joints, gradually increasing, and followed, after some hours or days, by fever.

3. The invasion is sudden—chills, a rapid rise of temperature, and inflammation of one or more joints.

When the disease is established there is a febrile movement, the pulse is full and rapid, the skin is hot and dry or bathed in acid perspiration, the tongue is coated, nausea and vomiting are often present, the bowels are constipated, the urine is diminished in quantity and loaded with urates, the intelligence is clear, but restlessness and sleeplessness are apt to be marked features.

One or more joints are inflamed, swollen, painful, and tender. The cases vary as to the severity of the pain, the intensity of the synovitis, the number of joints inflamed, and the way in which one joint after another is involved.

COMPLICATIONS: 1. *Hyperpyrexia.*—The disease begins mildly or severely. It runs its ordinary course for a few days and then the temperature rises rapidly, while the inflammation of the joints subsides. The sweating ceases, the tongue becomes brown and dry, the pulse is rapid and feeble, the breathing is rapid, the patients suffer from restlessness, sleeplessness, headache, hyperæsthesia of the skin, muscular twitchings, general convulsions, delirium, and coma. They usually die in a few days.

2. *Pericarditis and Endocarditis.*—These inflammations are especially common in young persons. They occur both in mild and severe cases. They usually follow the joint lesion, but may precede it, or even occur without it.

If a pericarditis is developed the temperature rises, the pulse is more rapid, there is præcordial pain, and more or less dyspnœa. The physical signs vary with the presence of fibrin alone, or of both fibrin and serum.

Some of the cases recover completely, others recover but with permanent pericardial adhesions, while still others die from the pericarditis.

If an endocarditis is developed there is a rise in the temperature, the patients look worse, the heart's action becomes



rapid, tumultuous, or irregular ; after one or two days a valvular murmur can be heard. After the patients have recovered, the valves which have been inflamed are left permanently damaged.

3. Complicating inflammations of the pia mater, pleura, or lung give their ordinary symptoms.

*Duration.*—The regular duration of an attack of acute articular rheumatism seems to be three weeks, but the disease may run its course within a few days ; it may last for months, or its course may be protracted by relapses.

*Terminations.*—Some of the patients recover completely, and never have another attack of the disease.

Others suffer from a number of attacks at short or long intervals.

In some the rheumatism runs its course, but one joint remains inflamed.

In some the convalescence is unusually prolonged, the patients remain feeble and anæmic, with stiff and tender joints.

In a considerable number the pericarditis or endocarditis lays the foundation of serious cardiac disease.

Death in the course of acute rheumatism is usually due to hyperpyrexia, pericarditis, endocarditis, pneumonia, pleurisy, or meningitis.

*Treatment.*—1. Of mild cases with inflammation of only one joint. These can often be very well managed by the application of continuous cold to the inflamed joint, with small doses of opium.

2. Of the regular well-marked cases. The majority of these are to be treated by the salicylate of soda, 10 to 20 grains every two hours ; the oil of wintergreen, 20 minims every two hours ; or salicin, 10 to 20 grains every two hours. The dose of these medicines is diminished as the disease subsides. The favorable effect of these remedies is seen in the improvement of the constitutional symptoms and the inflammation of the joints. They do not seem to shorten the duration of the disease or lessen the liability to complicating inflammations.

Some patients, however, are not at all benefited by the use of these drugs ; others get their poisonous instead of their medicinal effects.

There are fat, florid, overfed patients who, when they have an attack of acute rheumatism, are best treated by alkalies.

The patients are given 3 ij. of bicarbonate of potash in solu-

tion, with  $\frac{3}{4}$  j. of lemon-juice, every three hours for four days. After this they take 30 grains of bicarbonate of potash with 3 grains of quinine dissolved in lemon-juice three times a day. The bowels are to be kept open every day.

There is a group of cases characterized by excessive pain in the joints, without very much inflammation of them. The best remedies for these patients are phenacetine in 5-grain doses every two hours, antipyrine in 10- to 20-grain doses every two hours, or salol in 5-grain doses every two hours.

There are feeble and anæmic patients who, when they have an attack of acute rheumatism, are not benefited by any of the drugs which have been mentioned. They have to be treated by the iodide of potash, iron, quinine, and cod-liver oil.

Patients with the gouty diathesis, when they have acute rheumatism, often improve more rapidly if we combine colchicum with some of the drugs already mentioned.

Cases complicated by hyperpyrexia are to be treated by cold baths.

If the patients recover from their rheumatism but one joint remains inflamed; this joint is to be treated first by the application of cold, then by rest and pressure, and then by massage and passive motion.

The protracted convalescence is to be treated by change of climate, iron, quinine, and strychnine.

#### SUBACUTE RHEUMATISM.

In this form of rheumatism the invasion of the symptoms is gradual. The patients lose their appetite, the bowels are constipated, the tongue is coated, they become anæmic, feeble, and emaciated. A number of the joints are moderately inflamed, a little swollen, somewhat painful and tender. There may be a complicating endocarditis.

The disease is apt to last for a long time and to recur a number of times in the same patient. Its treatment is often very unsatisfactory.

We employ the salicylate of soda, antipyrine, phenacetine, iron, quinine, strychnine, the alkaline and sulphur mineral waters and baths, massage, and change of climate.

## MUSCULAR RHEUMATISM.

This is an affection of the muscles, apparently of inflammatory character, which causes them to become painful and tender. The muscles most frequently affected are those of the shoulder, neck, and back.

*Causes.*—The disease is especially common in adult life. The same person often suffers from a number of attacks. There is frequently a distinct history of exposure of the affected muscles to a draught of cold air. Similar conditions of the muscles are developed after over-exertion and as a complication of scarlet fever.

*Symptoms.*—The disease occurs in different degrees of severity.

1. One or more muscles, within a few hours, become painful on motion. If the muscles are not moved there is no pain. There is no constitutional disturbance, and the patients continue to feel well. The muscles remain in this condition for days or weeks. Hardly any treatment is necessary.

2. There is not only pain on motion in the affected muscles, but also pain when they are at rest, and this pain comes on in paroxysms which are exceedingly severe.

3. A large number of the muscles of the back are involved, the pain is severe, there is some rise of temperature, and the patients feel sick.

These more severe cases require treatment. We use as local applications mustard-plasters, hot fomentations, and the faradic current. The most efficient drugs seem to be phenacetine, antipyrine, or the iodide of potash and gelsemium given together.

## GONORRHŒAL RHEUMATISM.

This name is given to an inflammation of the joints and the tissues about them, which has nothing to do with real rheumatism, but is a specific inflammation accompanied with the growth of the same bacteria which are found with the specific urethritis.

There are two varieties of gonorrhœal rheumatism :

1. There is an inflammation of the tissues around the joints, with diffuse swelling, pain, tenderness, and redness of the skin. The ankle-joints are those most frequently involved, but the wrists and the other joints may be affected in the same way.

The inflammation is of acute or subacute character, and is apt to be long and tedious. It terminates in complete recovery, in stiffening of the joints, or in suppuration.

*Treatment.*—The inflammation is to be treated first by the continuous application of cold, then by rest and pressure, and finally by massage and passive motion.

2. There is a subacute synovitis, which involves most frequently one of the knee-joints. The joint is distended with serum, the inflammation may become purulent, and end in destruction of the joint.

*Treatment.*—Extension, pressure, or the opening and scraping of the joint are the methods of treatment.

### CHRONIC RHEUMATISM.

*Lesions.*—A chronic inflammation begins in the synovial membrane of one or more joints. The inflamed membrane becomes dense and thick, the fluid within the synovial sac is usually scanty and grumous, sometimes abundant and serous. The inflammation may extend to the capsules and ligaments of the joints, the articular cartilages, and the bones.

As complicating lesions we find chronic endocarditis, chronic endarteritis, pulmonary emphysema and bronchitis, and chronic nephritis.

*Causes.*—The disease belongs to adult life and to old age. It is especially common among the poor and among persons who are constantly exposed to all sorts of weather. In some persons the course of the chronic rheumatism is varied by attacks of acute rheumatism.

*Symptoms.*—The symptoms are principally the local symptoms of the inflamed joints. But from time to time there may be a little rise of temperature, and, after a while, digestive disturbances, loss of nutrition, and the symptoms belonging to the complicating lesions are developed.

The inflammation begins in one or more joints, often those of the hands and feet, continuing in these joints and then gradually extends to others, until in the worst cases nearly all the joints in the body are involved. The affected joints are tender and painful, swollen, sometimes fluctuating, sometimes stiff, sometimes creaking. The course of the inflammation is slow, with remissions and exacerbations.

The patients vary as to the position and number of the joints inflamed, and as to the extent of the changes in the joints.

The prognosis is always bad, but yet in some patients the disease never cripples them completely.

The only efficient treatment consists in the use of the natural mineral waters taken internally and as baths. The best waters seem to be those of Sharon, Richfield, Virginia, North Carolina, Arkansas, Wiesbaden, and Aix-les-Bains.

#### ARTHRITIS DEFORMANS.

*Synonyms.*—Rheumatoid Arthritis, Rheumatic Gout, Rhumatisme Nouveux, Chronic Rheumatism.

*Lesions.*—There is a chronic inflammation of the joints which from the outset, involves the cartilages, bones, and synovial membranes—a chronic pan-arthritis. The articular ends of the bones become thickened, flattened, and deformed by outgrowths of bone; the cartilages are softened, eroded, and destroyed; the synovial membranes are thickened. The joints become stiffened, deformed, sometimes dislocated.

*Causes.*—The cases in which the joints of the hands and feet are affected are most common in adult females belonging to the poorer classes. The form which involves the larger joints is most common in adult males. It is, however, possible for either form of the disease to occur in children.

*Symptoms.*—The arthritis comes on slowly and gradually. Usually the joints of the hands and feet are first involved, but any one of the larger joints may be the first affected. The natural tendency of the disease, as it advances, is to attack one joint after another.

The inflamed joints become painful and tender; then gradually stiffened, swollen, deformed, and dislocated. The corresponding muscles become contracted and atrophied. From time to time there may be exacerbations of the arthritis, with a rise of temperature.

The course of the disease is slow, but progressive. The patients become more and more helpless; digestive disturbances are developed; loss of flesh and strength and anæmia become more and more marked. The condition of the patient is rendered still worse by complicating lesions: Bronchitis, chronic nephritis, emphysema, and chronic endarteritis.

*The treatment* is very unsatisfactory. The only thing that seems to be of any service is the use of the natural mineral waters and baths, or the continued use of the iodide of potash.

### GOUT.

It is customary to describe acute gout, chronic gout, and irregular gout.

Our notions concerning the true nature of gout can hardly be considered as settled. But at the present time the views of Garrod are accepted as a probable theory.

Gouty inflammations of the joints are caused by the deposition of urate of soda in the articular cartilages. The functions of the kidneys are disturbed, and, after a time, they become the seat of a chronic diffuse nephritis with little or no exudation. During an attack of gout the blood becomes less alkaline, or there is an increase of uric acid in it, or there is a diminished excretion of uric acid by the kidneys.

*Lesions.*—The blood before and during an attack of gout contains an increased quantity of urate of soda. In chronic and irregular gout there is always an increase of urate of soda in the blood.

In the joints there is an acute or chronic inflammation, with a deposition of urate of soda in the articular cartilages. Deposits of urate of soda are also formed in the synovial membranes, the ligaments, the bursæ, the sheaths of tendons and of muscles, the cartilages of the ears, and under the skin.

The kidneys become the seat of a chronic nephritis with little or no exudation, but with a very large production of interstitial connective tissue.

Complicating lesions are of very frequent occurrence : Bronchitis, emphysema, endocarditis, endarteritis, eczema, and chronic catarrhal gastritis.

*Causes.*—The hereditary disposition to the disease is very marked. The gouty disposition may be acquired by the excessive use of nitrogenized food, of strong wines, and of malt liquors ; by a sedentary life ; by lead-poisoning ; and by insufficient nourishment. The disease is especially prevalent in temperate and damp climates.

As exciting causes of attacks of gout we recognize : Certain articles of food and drink, exposure to the weather, and injuries.

*Symptoms : 1. Of Acute Gout.*—An attack of acute gout is apt to come on during the night. There is sudden pain in one of the great-toe joints, chilliness, fever, restlessness, and sleeplessness. The next day the joint is swollen, the skin about it red, tense, and shining. The joint is extremely tender and painful. The urine is scanty, high-colored, and acid. Before and during the attack the quantity of uric acid in the urine is diminished ; after the attack it is increased. Such an attack lasts for days or weeks.

The first attack is usually, but not always, without premonition. The subsequent attacks are preceded by indigestion, constipation, flatulence, palpitation of the heart, bronchitis, irritability of temper. The urine is scanty and high-colored, or abundant and pale.

One of the great-toe joints is the joint most frequently inflamed ; next in order of frequency are the ankle, the instep, the outer side of the foot, the knee ; or several joints may be inflamed at the same time. After the inflammation has subsided the joint returns to its natural condition, or may be left a little stiff. The intensity of the inflammation of the joint varies very much in the different cases.

The height of the temperature is usually in proportion to the severity of the joint lesion.

Instead of the regular attacks of acute gout with inflammation of the joints, there may be acute attacks with little or no joint trouble, but with great prostration and symptoms referable to the stomach, heart, brain, or lungs.

In the stomach there is intense pain, nausea, and vomiting.

If the heart is affected there are pain, oppression, palpitation, feeble heart action, and sometimes syncope.

The brain symptoms are severe headache and delirium.

The pulmonary symptom is spasmodic asthma.

It is possible for a person to have only one attack of acute gout, but usually the attacks are repeated, and often become more frequent as the patient grows older.

*Treatment.*—During the attack the patient is to be kept quiet, on a light diet, with perhaps some opium to relieve the pain. The bowels are to be moved freely every day, and considerable quantities of fluid are to be drunk. The inflamed joint must be kept quiet and free from pressure.

The most efficient drug is colchicum, the wine of the root, or

the acetous extract. The dose at first is considerable, thirty minims to a drachm of the wine, one to two grains of the extract; later, these doses are diminished. The colchicum is usually combined with the salicylate of soda or the iodide of potash. The objection to the use of colchicum is its liability to produce vomiting and diarrhœa. Instead of colchicum, phenacetine in 5-grain doses every two hours will sometimes answer very well.

To prevent the recurrence of attacks the diet must be restricted by diminishing the ingestion of sugars, starches, and alcoholic drinks. Exercise and an out-of-door life are of much service. Any disturbances of the functions of the stomach, the liver, or the bowels should be remedied as far as possible. The occasional use of the mineral waters and baths is often of much service.

*Chronic Gout.*—The patients suffer from acute attacks at varying intervals, but between the attacks their health is not good. The joints are constantly somewhat inflamed, and become stiff and useless. Urate of soda is deposited in masses around the joints and in other parts of the body. Disturbances of digestion are troublesome and hard to relieve. The chronic nephritis, with its attendant symptoms, advances from year to year. The complicating bronchitis, emphysema, endocarditis, endarteritis, eczema, or diabetes add their symptoms. The patients grow slowly worse and, if not cut off by some intercurrent disease, finally die with the nephritis.

*Treatment.*—The indications for diet and treatment are the same as in acute gout. The natural mineral waters, especially those of Carlsbad, are of even greater service. The drugs most frequently used are colchicum in small doses, the iodide of potash, guaiac, the preparations of potash, soda, and lithia.

#### IRREGULAR GOUT.

A person is said to have irregular gout when he has the gouty diathesis and suffers from one or more of the complications of gout.

There are many morbid conditions, none of them confined to gouty persons, which may be caused in this way by the gouty disposition :

In the stomach—gastric dyspepsia and catarrhal gastritis.



In the liver—functional disturbances with nervous symptoms.

In the heart—palpitation and disturbances of sensation.

In the lungs—bronchitis.

In the eyes—iritis.

In the skin—eczema.

In the urine—an excess of urates, glycosuria, and diabetes.

In the kidneys—chronic diffuse nephritis.

In the arteries—chronic endarteritis, inflammation and rupture of the cerebral arteries.

In the joints of the fingers and toes—a gradual thickening of the ends of the bones without pain.

*The treatment* consists in the use of remedial measures appropriate to each one of these morbid conditions, and at the same time the general management of the gouty disposition.

## THE INFECTIOUS DISEASES.

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It is now generally believed that a large number of diseases and inflammations are caused by the presence and growth of micro-organisms. The organisms which produce suppuration, erysipelas, lobar pneumonia, gonorrhœa, anthrax, tuberculosis, tetanus, diphtheria, cholera, and typhoid fever have been isolated, cultivated, inoculated, and studied in many different ways. In many other diseases the existence of such organisms is probable, but has not yet been demonstrated.

These organisms seem to do harm in several different ways :

1. The organisms may act as local irritants, and by their presence in the tissues set up about them an exudative, productive, or necrotic inflammation. The tubercle bacilli can in this way set up a productive inflammation even when they are dead.

2. The organisms when introduced into the tissues do nothing unless those tissues are by some other cause inflamed. The organisms then grow in these inflamed tissues and modify the character of the inflammation.

3. The organisms introduced into one part of the body and growing there, may find their way into distant parts of the body and set up fresh foci of inflammation, but without constitutional infection. We see this in gonorrhœal rheumatism.

4. The organisms when introduced into one part of the body rapidly multiply and are soon found in large numbers in the blood and tissues throughout the body. They seem to cause constitutional symptoms partly by their numbers, partly by the chemical products evolved by their growth. Anthrax is one of the best examples of this.

5. The organisms when introduced into one part of the body grow and multiply there. They remain confined to this locality and are not found in the blood or in other parts of the body.

They produce at the locality where they grow poisonous chemical products which affect, more or less intensely, the entire body, and give rise to correspondingly severe constitutional symptoms. We see this in ordinary suppuration, in lobar pneumonia, and in diphtheria.

6. It has been found, in the case of some of these micro-organisms, that the poisonous chemical products evolved by their growth can confer upon animals an immunity, either against the growth of the same micro-organism or against the effects of its chemical poison.

It is in this way that it is hoped that, in the future, we will be able to arrest or modify the course of infectious diseases in the human being.

7. These micro-organisms can be conveyed into the human body by inoculation, by inhalation, or by swallowing. As a rule, each micro-organism has its preference, and habitually infects the body in one of these ways rather than in another. The cocci which produce abscesses, or erysipelas, or tetanus are usually inoculated; the bacilli which produce pneumonia are usually inhaled; those which cause typhoid fever or cholera are swallowed.

8. In many of the infectious diseases there is always present an inflammation of the skin, of the mucous membranes, or of the viscera, which is so constant that we call it the characteristic lesion of the disease. The micro-organisms are regularly found most constantly, and in the largest numbers, in these characteristic local lesions.

9. It is found that while some persons are susceptible to the poisons of the infectious diseases, other persons enjoy an immunity from the action of these poisons. It is also found that in some of these diseases one attack protects against subsequent attacks of the same disease. Temporary conditions of atmosphere, water, soil, and uncleanness favor the development of these diseases. Some of the infectious diseases occur only from time to time in the form of epidemics.

#### EPIDEMIC CEREBRO-SPINAL MENINGITIS.

*Definition.*—An infectious disease of which the characteristic lesion is an acute inflammation of the pia mater of the brain and of the spinal cord.

*Synonyms.*—Spotted fever; malignant purpura; typhus petechialis; typhoid meningitis.

*History.*—The first unquestionable descriptions of the disease are those given by Vieusseux and Mathey of an epidemic which occurred in Geneva in 1805. Its distinctive features were an abrupt attack during the night, vomiting, severe headache, rigidity of the spine, difficult deglutition, convulsions, petechiæ, death in from twelve hours to five days. After death a gelatinous or puriform exudation was found on the surface of the brain and of the medulla oblongata. Between the years 1806 and 1816 there were localized epidemics in Germany, Holland, France, England, and the United States. Then there was an interval of six years during which the disease disappeared. In 1822-23 there were small epidemics in France and in Connecticut. Then, after another interval of five years, it reappeared in 1828 in Ohio, two years later in England, and three years later in Naples.

After four years of quiescence the disease entered upon a wider career, which was almost uninterrupted from 1837 to 1850, and extended over a large part of Europe and of the United States.

Between 1850 and 1854 the disease again disappeared. Then there was a very fatal epidemic in Sweden, which lasted until 1861. In 1856-57 there were epidemics at different places in the United States. From 1860 to 1865 the disease was seen in Holland, Portugal, and Germany. From 1861 to 1872 there were epidemics in many parts of the United States, the disease prevailing in New York in 1872-73.

*Etiology.*—It is evident, from the history of the disease, that it is characteristic of it to appear and disappear during periods of years at places widely separated from each other. Its greatest prevalence and mortality have been in the northern portions of the temperate zone in Europe and America, and during the cool seasons of the year.

No locality seems to be exempt from the disease. It is seen in cities, villages, isolated houses in the country, barracks, prisons, and work-houses, without reference to soil or elevation. While all ages are liable to the disease, the greatest liability is among children and young adults.

It is generally believed that the poison of the disease is not transmitted from person to person, but is communicated to a number of persons at the same time.

The micro-organisms which seem to belong most constantly to the disease, being found regularly in the inflamed pia mater, are the pneumococci of Fränkel, the same organisms which are found in pneumonia, pleurisy, pericarditis, otitis, peritonitis, and in the saliva and nasal secretions of healthy persons. It is very difficult to understand how a disease which occurs principally in epidemics separated from each other by intervals of years, should be caused by one of the most common of the bacteria.

*Lesions.*—The characteristic lesion of the disease is an acute inflammation of the pia mater of the brain and spinal cord. In the great majority of cases this is a simple exudative inflammation, with an infiltration of the pia mater with serum, fibrin, and pus, these products accumulating in the largest quantity at the base of the brain and on the posterior surface of the cord. The lateral ventricles contain a little purulent serum. In children the quantity of purulent serum in the lateral ventricles is often so large as to distend them.

In a few cases there is no fibrin, serum, or pus, nothing but an increase of cells in the pia mater.

In a few cases it is said that the pia mater is congested, while no exudate is present.

*Symptoms.*—The different epidemics of the disease have varied as to whether the invasion was sudden, or preceded by a prodromic period.

If the prodromic period exists, it is characterized by general malaise, headache, pains in the bones, loss of appetite, conjunctivitis, chilliness, and a slight rise of temperature.

When the invasion is sudden, it is marked by one or more of the following symptoms: Chills, a rise in temperature, headache, vomiting, tenderness and contraction of the muscles of the back of the neck, irregular pains in different parts of the body, conjunctivitis, restlessness, sleeplessness or delirium, and prostration.

After the disease is fairly established the following are the regular symptoms: Pain in the head—usually frontal, sometimes occipital or general—is regularly severe and constant throughout the disease. The suffering is very great, so that the patients complain of it most bitterly, and even after they have become partially unconscious, from time to time they cry out with the pain. Exceptionally the pain is but moderate and intermits from time to time.

Pain, tenderness, and contraction of the muscles of the back of the neck, or also of the muscles along the vertebral column, are often present. The head alone is drawn back, or the entire body is bent back and rigid. Any attempt to move the contracted muscles gives intense pain.

There may be severe pain along the course of the spinal nerves, pain which moves rapidly from one part of the body to another. With this pain there may also be hyperæsthesia of the skin. Later in the disease the pain and hyperæsthesia are succeeded by numbness and insensibility.

There may be contractions of groups of muscles, especially of the muscles of the face, or general convulsions; the latter is especially common in children.

Instead of involuntary contractions of the muscles, there may be paralysees either of the muscles of deglutition, or of those of one of the extremities, or of those of a large part of the body. Ptosis, strabismus, and paralysis of the bladder and rectum occur quite frequently.

The eyes are liable to a variety of inflammations—conjunctivitis, keratitis, or choroiditis. Independently of inflammation, there is at first photophobia with contracted pupils, later insensibility to light with dilated pupils.

The hearing is at first unnaturally acute and painful, later it is dulled or lost. In some epidemics a large number of patients suffer from inflammations of the ear, which leave behind permanent deafness.

Restlessness, sleeplessness, delirium, stupor, and coma are present in various degrees, either early or late in the disease, or throughout its entire course. The delirium is mild, or active, or so violent as to resemble acute mania. The establishment of well-marked delirium and stupor alternating with each other, one or the other predominating as the disease goes on, belongs to the severe cases.

Vomiting is a very common symptom at the beginning of the disease. It may be so frequent and distressing as to constitute for a time the most important symptom.

The bowels are usually constipated, but during the later stages of the disease there may be diarrhœa and involuntary evacuations.

The tongue is at first moist and coated, later dry and cracked, if the disease is of severe type.

In some epidemics it is reported that the joints were inflamed. Sometimes they are swollen, red, and tender ; or red and painful without any swelling ; or swollen and painful without redness. The joints usually attacked are the knee, elbow, wrist, and the small joints of the fingers and toes.

The characteristic pulse of the disease is one which is slow in proportion to the height of the temperatures. Pulses of 60 and 70 to the minute are quite common, and ones even as slow as 27 are recorded. But exceptions to this rule are not infrequent, and in the severe cases the pulse regularly becomes more rapid as the disease goes on.

In the accounts of the earlier epidemics of the disease, when no thermometer was used, it is frequently stated that there was no fever. Since the introduction of the thermometer it has been found that there is a rise of temperature, often as high as 104° F. But the curve of temperature is very irregular and not in correspondence with the progress of the disease. A patient may be doing quite as badly with a temperature of 99° F. as with one of 104° F.

Eruptions of the skin are present in some epidemics, absent in others. They have been observed more frequently in the epidemics in America than in those in Europe. When the disease first appeared in New England a large proportion of the cases exhibited petechial eruptions and ecchymotic spots, whence the disease received the name of spotted fever. There have been observed in different epidemics ecchymoses in the skin, petechiæ, erythema, roseola, urticaria, and pemphigus.

The severity and mortality of the disease has varied in the different epidemics. In some the disease has been of most malignant type, the mortality as high as seventy-five per cent., and many of the cases dying within a few hours. In others the mortality has not been over twenty per cent., and the duration of the disease has been from one to four weeks.

In children the invasion of the disease is often marked by convulsions, which may afterward be frequently repeated. The temperature is high and the pulse rapid throughout the disease. During most of the time the children are in a condition of stupor alternating with restlessness.

In the regular cases there may be a temporary cessation of the symptoms for several hours, or even for two or three days, and then they go on as before.

The disease may be complicated by bronchitis, pneumonia, pleurisy, pericarditis, or endocarditis.

During the past few years I have seen in New York cases of cerebro-spinal meningitis which have run an atypical course.

The symptoms have come on gradually, some of the patients not in bed until after several days, none of them looking very sick for the first one or two weeks.

In some of the patients the first symptom has been a headache like that of malaria or of syphilis.

In others, besides the headache, there have also been loss of appetite, vomiting, photophobia, drowsiness, sleeplessness, tenderness over the back of the neck, or an eruption like that of typhoid.

In still others there has been no headache, only prostration, sleeplessness, and a little fever.

For one or two weeks, or even longer, the patients do not look very sick. They make you think of malaria, or syphilis, or a mild typhoid. But they do not get any better, they lose flesh and strength, develop alternating stupor and delirium, and finally die after from four to eight weeks.

The temperature is seldom over 102° F., often down to 99° or even 97° F.

The pulse is between 70 and 120. After death, in most of the patients the lesions found have been those of cellular meningitis, but in one case there was a large exudation of fibrin and pus at the base of the brain.

*Sequelæ.*—After the subsidence of the meningitis the inflammation of the lateral ventricles may continue. The patients begin to improve, but soon fall back. There is progressive emaciation, an irregular febrile movement, alternating stupor and delirium, and after several weeks the patients die.

Other sequelæ are: blindness, deafness, mental feebleness, aphasia, anæmia, hysterical symptoms, disordered menstruation, paralyses of groups of muscles, and chronic endocarditis.

*The prognosis* of the disease varies in different epidemics, regularly worse during the commencement and height of each epidemic.

*Treatment.*—There are two main indications for treatment—the meningitis and the general condition of the patient.

For the meningitis we abstract blood from the temples and the nape of the neck, apply continuous cold to the head, and ad-



minister repeated small doses of calomel or the sulphate of magnesia. The iodide of potash and ergot are also used for the same purpose. These measures are most likely to be efficacious during the early days of the meningitis.

For the headache, restlessness, sleeplessness, and delirium we give the bromides, chloral hydrate, or opium.

The temperature is seldom so high as to need any attention.

The heart's action may be so feeble as to require the use of alcoholic stimulants, digitalis, or strophanthus.

Excessive vomiting can be relieved by feeding the patients with small quantities of milk to which oxalate of cerium, bicarbonate of soda, or sulphonal has been added.

The bowels should be kept open.

#### DIPHTHERIA.

The name diphtheria, first employed by Bretonneau in 1820, has been used since that time to designate the inflammations accompanied by the formation of a false membrane, especially when such an inflammation occurs in the throat, nose, or larynx. For many years the true character of the disease was not understood, but more recently bacteriology has shown us that of these inflammations, with the formation of a false membrane, there are several distinct forms. We, therefore, now describe separately true diphtheria and false diphtheria.

#### STREPTOCOCCUS DIPHTHERIA.

*Etiology.*—Streptococci are micro-organisms which are found in rooms and dwellings, especially if these rooms are overcrowded and unclean. They are found in the secretions of the nose, mouth, and vagina in healthy persons. They grow and thrive in inflamed tissues, and increase the severity of the inflammation. They can be transported by the blood and lodged as infectious emboli in distant parts of the body. They vary greatly as to the intensity of the chemical poison produced by their growth, and the consequent severity of the constitutional symptoms. They are found in erysipelas, suppurating inflammations, pleurisy, malignant endocarditis, puerperal septicæmia, and in inflammations caused by other bacteria.

Streptococcus diphtheria is caused by the inflammations of

the nose, throat, and larynx which accompany measles and scarlet fever, especially if these diseases occur in an asylum where the rooms are full of streptococci. It also occurs in isolated cases, or in groups of persons in whom the throat becomes inflamed from any cause. It is much more common in children than in adults. It would seem probable that it is not communicated from person to person, but that the infection comes from the streptococci which are so frequently present in the air.

*Lesions.*—There is a croupous inflammation of the mucous membrane of the tonsils, pharynx, nose, or larynx. The inflamed mucous membrane is congested, swollen, infiltrated with fibrin, pus, and coated with a false membrane composed of fibrin, pus, and dead epithelium. The streptococci are found in the false membrane and the mucous membrane beneath it. The inflammation often travels down the bronchi and produces a broncho-pneumonia. There does not seem to be the same disposition to acute degeneration of the viscera and of the nerves that exists in the Klebs-Löffler diphtheria.

*Symptoms.*—The symptoms are especially those belonging to the local inflammation. The obstruction in the nose, the pain in the throat, the cough and dyspnoea of the laryngitis are regularly present. The principal constitutional symptoms are the rise of temperature and the disturbances that go with it. Otherwise there is little evidence of general poisoning. The principal dangers are from the laryngitis and the broncho-pneumonia. If the patient escapes these the probabilities of recovery are good. The disease runs its course, exclusive of the broncho-pneumonia, within a week or two weeks. When only the tonsils or pharynx are involved the patients, as a rule, are not at any time very sick.

There are, however, cases of streptococcus diphtheria which are much more serious. There is necrosis of the tissues adjacent to the inflamed mucous membrane. The patients give all the evidences of septic poisoning and are very apt to die. These bad cases are especially frequent as complications of scarlatina.

*Treatment.*—It is not considered necessary to isolate patients who have streptococcus diphtheria. But the occurrence of any number of such cases in one building means that the building needs a thorough cleaning.

The treatment of the inflammation is local. It consists in the frequent irrigation of the inflamed mucous membrane with solutions of bichloride of mercury or peroxide of hydrogen.

## TRUE DIPHTHERIA.

*Etiology.*—The Klebs-Löffler bacillus is about the length of the tubercle bacillus. It does not multiply, or does so very sluggishly, at a point below 64° F. It is killed by exposure for ten minutes to a temperature of 136° F. It remains alive outside of the body, in spite of drying and unfavorable influences, for many months. It develops only locally at the seat of infection, and does not invade the tissues or the circulation. It is found in the false membrane, and not in the mucous membrane beneath it.

The bacilli are conveyed from one person to another in the portions of false membrane and saliva discharged from the mouth and nose. These infectious particles may become lodged about the person and clothing of the patient, and in the bedding, furniture, etc. The bacilli are probably not present in the breath of the patient. As they are not destroyed by drying, after they have been lodged in the clothing, etc., they can float in the air and be inhaled.

The bacilli, either when growing in the false membrane, or when cultivated outside the body, produce a toxic substance of the most virulent character. This substance, when inoculated in animals, produces all of the changes in the body which follow the inoculation of pure cultures of the bacilli, with the exception of the false membrane.

True diphtheria, therefore, is a disease communicated from person to person by a specific bacillus which grows in the false membrane. The inoculation is ordinarily effected in the mucous membrane of the throat, nose, or larynx. It is probable that an antecedent inflammation of these mucous membranes favors the inoculation. Children are more susceptible than are adults. The disease prevails more extensively during some years than during others.

The disease begins as a local inflammation, and the constitutional symptoms and secondary lesions are due to a poison which is formed by the growth of the bacteria.

The Löffler bacilli usually disappear from the throat within three days after the complete disappearance of the exudation, but it is not rare to find them for as long as seven days, and cases are recorded in which the bacilli were found for nine weeks after the disappearance of all membrane.

It is well established that virulent and non-virulent diphtheria bacilli are found in the throats of healthy persons, and that this is more apt to occur if they have been exposed to diphtheria. The same thing is true of diphtheria bacilli as of streptococci and pneumococci, that, although they are capable of producing virulent poisons, they can also grow in the human body without doing any harm.

It is to be remembered that in diphtheria the infection is not with diphtheria bacilli alone, but that it is a mixed infection, as either streptococci, or staphylococci, or both, are also usually present.

*Lesions.*—The mucous membranes ordinarily inflamed are those of the tonsils, pharynx, nose, larynx, trachea, and bronchi. The inflammation begins in one of these situations, and then is apt to extend to the others. The inflamed mucous membrane is congested, swollen, infiltrated with serum, fibrin, and pus; its surface is coated with a false membrane composed of fibrin, pus, and necrotic epithelium. The necrosis involves the epithelium alone, or also portions of the stroma of the inflamed mucous membrane. In the false membrane are found the Klebs-Löffler bacilli, and in addition a variety of other micro-organisms. The cases vary much as to the intensity of the inflammation and the quantity of false membrane.

Beside the regular cases of croupous inflammation caused by the diphtheria bacillus, there are examples of ordinary catarrhal inflammation of the mucous membrane of the pharynx and tonsils also accompanied by the growth of the diphtheria bacillus. It is customary to include these cases under the name of diphtheria.

The inflammation may extend down the trachea and produce a broncho-pneumonia.

The adjacent lymphatic glands are often inflamed and swollen, with more or less necrosis of their cells.

The kidneys become the seat of acute degeneration, acute exudative nephritis, or acute diffuse nephritis.

The nerves may become the seat of acute degeneration.

*The symptoms* of diphtheria are both local and constitutional.

The appearance of the inflamed throat varies with the exact character of the inflammation. The mucous membrane is swollen, congested, red, coated here and there with small patches of false membrane, or covered with a very thin membrane, or very much

swollen and covered everywhere with a thick membrane. The inflammation remains confined to the situation where it began, or it extends. It regularly runs its course by the end of seven days, then the swelling diminishes, the mucous glands pour out an increased quantity of mucus, and the false membranes become detached.

When the quantity of false membrane is great it undergoes putrefaction, and the patient's breath has a foul smell.

The sore throat is regularly, but not always, accompanied by pain and difficulty in swallowing.

With the inflammation of the tonsils and pharynx there is often more or less swelling of the lymphatic and salivary glands in the neck.

If the inflammation begins in the nose, it gives at first few symptoms and is easily overlooked.

The inflammation may begin in the larynx, or it may extend to it after from five to seven days. It is attended with laryngeal cough, voice, and dyspnoea. Such a laryngitis is always dangerous, especially in children.

Broncho-pneumonia, either with or without laryngitis, may be developed from the fifth to the seventh day of the disease. The physical signs are the ordinary ones of broncho-pneumonia. With this complication all of the patients at once look decidedly worse, but only in some of them are there pulmonary symptoms. This lesion is a very fatal one.

The invasion of the disease is marked in some cases only by the sore throat or the laryngitis; in other cases by chills, a rapid rise of temperature, or general convulsions.

A febrile movement may be absent throughout the disease; may be one of the first symptoms and continue nearly up to the time of the patient's death; may be present at first and then subside, although the other symptoms continue; may be absent at first and not appear until several days after the development of the throat lesion. In some of the worst cases there is little or no fever.

Vomiting may be a troublesome symptom at any time in the disease.

Diarrhoea is occasionally present.

The heart's action and the radial pulse become rapid and feeble according to the severity of the disease.

The patients are liable during the disease, and during conva-

lescence, even as late as three or four weeks after the invasion of the disease, to attacks of heart failure. These attacks of heart failure occur in one of two ways: The patient may be in his ordinary condition, when the heart will suddenly stop and death is almost instantaneous. Or the heart's action becomes very rapid and feeble, with cardiac dyspnœa and venous congestion; it continues to get worse for a number of hours until the patient dies.

In most of the patients the kidneys are diseased in one of three ways.

There may be acute degeneration of the kidney. The urine is diminished, its specific gravity remains normal, it contains little or no albumin. This change in the kidneys seems to add little to the dangers of the diphtheria.

There may be acute exudative nephritis. The urine is diminished in quantity, its specific gravity remains normal, it contains much albumin and many casts. Such a complicating nephritis may prove fatal.

There may be acute diffuse nephritis. This occurs during convalescence. The symptoms are acute or subacute. The nephritis regularly persists and becomes chronic.

*Course of the Disease.*—1. The Malignant Cases. (a) The invasion of the disease is acute. It may begin with general convulsions. The temperature rises rapidly. The inflammation of the throat is severe, extensive, and rapidly extends, with great swelling and a large production of false membrane. The salivary and lymphatic glands in the neck are usually swollen. The patients rapidly become worse, stupor and delirium are developed, and death follows in from three to five days.

(b) The invasion of the disease is slow and gradual. The child is at first ailing, but without fever or pain in the throat. The neck is swollen, the throat is congested, with little or no visible false membrane, the skin is pale and cool, a dirt-brown fluid exudes from the mouth and nose. The patient dies in collapse in from three days to two weeks.

2. The Ordinary Cases. One tonsil is inflamed with a small patch of membrane on it. Fever is present or absent. The inflammation and the false membrane gradually spread over the rest of the throat, with more or less fever, vomiting, and prostration. By the seventh day the inflammation of the throat subsides, the membrane begins to come away, and there is an increased production of mucus. If the larynx or bronchi are not

involved the patients get better and are convalescent by the end of three weeks. The complicating laryngitis and broncho-pneumonia are liable to be developed between the fifth and tenth day, with their characteristic symptoms. The cases vary as to the predominance of the throat or the constitutional symptoms, and as to the sthenic or asthenic type of the disease. All the patients are liable to attacks of heart failure and to sequelæ.

3. Cases in which both the throat lesions and the constitutional symptoms are but slightly developed. These patients do very well, but they are a source of danger to the community; for they are not confined to bed, nor to the house, and so are likely to transmit the disease to other persons.

4. Cases which at first have mild local and constitutional symptoms. But after a few days the inflammation of the throat suddenly becomes worse and the patients much prostrated. Such patients often do badly. The duration of the disease has to be dated from the day when the patients suddenly become worse.

5. Cases in which the larynx is first inflamed. This is especially common in young children. The first symptoms are those of the laryngitis, the symptoms of the diphtheria are developed later. This is a very fatal form of the disease.

6. Cases in which the disease is protracted. The inflammation of the throat runs its course within seven days, and the false membrane begins to come away. But after a day or so a fresh attack of inflammation, with a new false membrane, are developed, and this may be repeated several times; although these patients are sick for a long time, most of them eventually recover.

7. Cases which behave like ordinary cases of catarrhal pharyngitis or tonsillitis, but yet there are virulent bacilli on the surface of the mucous membrane or in the crypts of the tonsils. The diagnosis has to be made by the bacteriological examination.

The convalescence after diphtheria is slow, and it is often months before the patients fairly regain their health.

*Sequelæ* are of frequent occurrence. There may be paralyses of groups of muscles, apparently due to inflammation or degeneration of the peripheral nerves. The muscles usually paralyzed are those of the soft palate, the pharynx, the larynx, and the eye, the muscles which control respiration, and the muscles of the arms and legs. These paralyses, as a rule, after lasting for weeks

or months, disappear. They are not dangerous to life unless they involve the muscles of respiration.

A post diphtheritic subacute productive nephritis may be developed.

There may be sudden or prolonged heart failure.

*The prognosis* of the streptococcus diphtheria is very much better than that of true diphtheria. But yet, if the larynx is involved, if there is broncho-pneumonia, or if the patients are very young, the danger is great.

In true diphtheria the prognosis varies with the intensity of the poison produced by the bacilli, with the presence or absence of laryngitis and broncho-pneumonia, and with the age of the patient.

*Prophylaxis.*—The prevention of streptococcus diphtheria is of especial importance in children's hospitals and asylums. By sufficient care in keeping the rooms and wards clean, the ordinary cases of measles, scarlet fever, and whooping-cough will run their course without any complicating diphtheria. In the case of persons suffering from the disease the discharges from the mouth and nose should all be received into germicide solutions of sufficient strength to destroy the streptococci.

In true diphtheria the danger lies in the false membrane. The most minute care, therefore, is necessary that all the discharges of the mouth and nose should be received into germicide solutions. Even with the greatest precautions, fragments of the membrane become lodged in the bedding, clothing, carpets, etc. All the clothing, therefore, should be steamed and washed, the mattresses and pillows destroyed, the carpets taken up and cleansed, the furniture and room washed with bichloride solution and repainted.

*Treatment.*—The first thing is to determine whether the patient is suffering from streptococcus diphtheria or from true diphtheria. This is done most certainly by a bacteriological examination. For this purpose we are, in New York, supplied by the Board of Health with a culture outfit which consists of a small wooden box containing a tube of blood serum and a tube containing a swab.

The patient should be placed in a good light, and, if a child, properly held. The swab is removed from its tube, and while the tongue is depressed with a spoon it is passed into the pharynx (if possible, without touching the tongue), and is rubbed



gently but firmly against any visible membrane on the tonsils or in the pharynx, and then, without laying the swab down, it is immediately inserted in the blood-serum tube, and the portion which has been previously in contact with the exudate is rubbed a number of times back and forth over the whole surface of the serum. This should be thoroughly done, but it is to be gently done, so as not to break the surface of the serum. The swab is replaced in its tube, and both tubes, their cotton plugs having been inserted, are returned to the box and sent to the collecting station. The blank forms of report which accompany each outfit should be completely filled out and forwarded to the station with the tubes.

Where there is no visible membrane (it may be present in the nose or pharynx) the swab should be thoroughly rubbed over the mucous membrane of the pharynx and tonsils, and in nasal cases, when possible, a culture should also be made from the nose. In little children, care should be taken not to use the swab when the throat contains food or vomited matter, as then the bacterial examination is rendered more difficult. Under no conditions should any attempt be made to collect the material shortly after the application of disinfectants (especially solutions of corrosive sublimate) to the throat. If any of these instructions have not been carried out, the fact should be carefully noted on the record blank.

The culture-tubes which have been inoculated as described above, are kept in an incubator at  $37^{\circ}$  C. for twelve hours, and are then ready for examination. On inspection, it will be seen the surface of the blood serum is dotted with very numerous colonies, which are just visible. At this time no diagnosis can be made from simple inspection (if, however, the serum is found liquefied, or shows other evidences of contamination, the examination will probably be unsatisfactory). A microscopical preparation is now made by placing a tiny drop of water upon a clean cover-glass, and then a platinum needle is inserted in the tube, and quite a large number of colonies are swept with it from the surface of the culture medium. The bacteria adherent to the needle are washed off in the drop of water previously placed on the cover-glass and smeared over its surface. The bacteria on the glass are then allowed to dry in the air. The cover-glass is then passed quickly through the flame of a Bunsen burner or alcohol lamp three times in the usual way, covered with a few

drops of Löffler's solution of alkaline methyl blue, and left without heating for ten minutes. It is then rinsed off in clean water, dried, and mounted in balsam.

In the great majority of cases, one of two pictures will be seen with the  $\frac{1}{12}$  oil immersion lens; either an enormous number of characteristic Löffler bacilli with a moderate number of cocci or a pure culture of cocci, mostly in pairs or short chains. In a few cases there will be an approximately even mixture of Löffler bacilli and cocci, and in others a great excess of cocci. Besides these there will be occasionally met preparations in which, with the cocci, there are mingled bacilli more or less resembling the Löffler bacilli. These bacilli, which are pseudo-diphtheria bacilli, are especially frequent in cultures from the nose.

In not more than one case in twenty will there be any serious difficulty in making the diagnosis, if the serum-tube has been properly inoculated. In such a case, another culture must be made.

All treatment is most efficacious during the first three days of diphtheria. The local treatment of the throat and nose is best carried out by frequent irrigations with solutions of bichloride of mercury in the strength of from 1 to 4,000, to 1 to 10,000, or of a three per cent. solution of peroxide of hydrogen.

The best local treatment for the larynx seems to be by the inhalation of the fumes of calomel. But if the larynx is much obstructed either intubation or tracheotomy becomes necessary to prevent suffocation.

To counteract the effects of the poisoning it has been customary to use alcoholic stimulants in considerable quantities, and the tincture of the chloride of iron in doses of from five to twenty minims every two hours.

Since the year 1893 the treatment of diphtheria by antitoxin has been carried on in many different places and with large numbers of patients. It is the belief of those best qualified to judge that by this treatment the mortality from diphtheria can be very much diminished.

The so-called diphtheria antitoxin is prepared in the following way: Pure cultures of the diphtheria bacillus are allowed to grow in bouillon. As they grow the toxins of diphtheria are formed and held in solution in the bouillon. If the bouillon is filtered we have a fluid containing toxins. To determine the strength of this solution we employ injections into guinea-pigs.

When the strength of the toxin solution is such that 0.1 c.c. kills a guinea-pig of about 250 grammes weight within forty-eight hours, it is called normal or standard toxin. This is the solution of toxins which is used to immunize animals. The animal which has been found to be the most convenient for this purpose is the horse. In order to immunize a horse we begin with subcutaneous injections of half a cubic centimetre of standard toxin. The injections are repeated at intervals with gradually increased quantities of toxin until the animal tolerates as much as 200 c.c. at one injection. The time required to acquire such a tolerance is about three months, and the horse is then said to be immunized.

The next thing is to obtain the blood serum of the immunized animal. For this purpose, from 6 to 8 litres of blood are drawn off from the jugular vein. This blood is allowed to clot in a cool place, the serum is drawn off, a little carbolic acid or other antiseptic is added, and we have the antitoxin serum. Before it is used the strength of each batch of the antitoxin serum has to be determined. This is done by finding out how large a quantity of antitoxin will render the standard toxin harmless to guinea-pigs. If 0.1 c.c. of antitoxin renders 1 c.c. of normal toxin harmless to a guinea-pig weighing 250 grammes, it is called normal antitoxin, and 1 c.c. of normal antitoxin is called an antitoxin unit. The serum employed in practice is made so strong that 1 c.c. contains the strength of many antitoxin units. Experience has shown that to use antitoxin for the cure of diphtheria it must be employed within the first three days of the disease. The quantity injected at one time varies between 600 and 1,500 normal antitoxin units in about 10 c.c. of serum, according to the age of the patient and the severity of the disease.

In the favorable cases improvement begins within twenty-four hours after the injection. The local inflammation subsides and the membrane begins to become detached; the fever and other constitutional symptoms also become less severe.

How far the sequelæ of diphtheria-nephritis—heart failure and paralysis—are favorably affected by antitoxin is not exactly determined.

The antitoxin itself may produce some poisonous effects. Inflammations of the skin like those of erythema, urticaria, measles, and scarlatina are of not infrequent occurrence. While in some

patients fever, prostration, with pain and swelling of the joints, are observed.

Throughout the disease attention to the nursing and feeding is of the greatest importance.

The convalescence is often tedious, and it may be months before the patient's health is fully restored.

#### EPIDEMIC INFLUENZA.

*Synonyms.*—Epidemic Catarrhal Fever ; La Grippe.

*Definition.*—An infectious disease, caused by the presence and growth of the influenza bacillus, characterized by a rise of temperature, prostration, pains and inflammations of different parts of the body. The parts of the body which may be inflamed are : the pia mater, the peripheral nerves, the mucous membranes of the nose, throat, larynx, bronchi, stomach, and colon, the lungs, the pleura, the pericardium, and the skin.

*History and Causes* (Ziemssen).—The history of influenza can be traced back with certainty only to the beginning of the sixteenth century. With the year 1510 begins a series of epidemics the wide distribution of which has been reached by no other acute infectious disease. Up to the year 1870, more than ninety epidemics have been described, which generally extended over whole countries, or whole quarters of the earth. As a rule, the epidemics advance from the east and northeast toward the west. Sometimes they occur simultaneously at several different parts of the earth. The epidemics lasted, as a rule, only four to six weeks ; only exceptionally, as in Paris in 1831, have they lasted for nine or ten months. Wherever the epidemics prevail, very large numbers of the population are attacked. There is no special liability or immunity by reason of age, sex, constitution, or condition of life ; but in some epidemics children are exempt. The influenza, as such, is not usually fatal, but while it prevails the general death-rate is increased. Climate and atmospheric influences seem to exert no effect. One attack of the disease does not protect against subsequent attacks.

*Lesions.*—The bacillus which is believed to be characteristic of influenza has been found in the sputum, the lungs, and the blood. The inflammations which it causes are of exudative type.

The pia mater of the brain and cord is infiltrated with serum, fibrin, and pus.

The sheaths of the nerves are swollen. The changes in the mucous membranes of the frontal sinuses, nose, throat, and larynx are those of an ordinary acute catarrhal inflammation. The bronchitis is characterized by an unusually large production of mucus, and of pus-cells with considerable disposition to bleeding.

In the lungs the pneumonia is of the lobar form, but with a tendency to excessive congestion and incomplete hepatization.

The pleurisy and the pericarditis may be with fibrin alone, or with fibrin and serum, or with fibrin, serum, and pus.

The catarrhal gastritis is sometimes of severe type.

The colitis may involve either the lower or upper part of the colon.

In any one case either one or several of these inflammations may be present.

*Symptoms.*—Few diseases offer such a variety in their symptoms as does influenza. Each form of the disease closely resembles one of the ordinary inflammations or diseases, so that errors in diagnosis are easy. Often we would be unable to determine the character of the attack if it were not for our knowledge that an epidemic of influenza is prevailing. The ordinary forms of the disease are as follows:

1. Cases characterized by fever alone. These patients complain little, or not at all, of headache or of pains, nor do they have symptoms referable to any of the viscera. But every day the temperature ranges between  $99^{\circ}$  and  $103^{\circ}$  F., lower in the morning, higher in the evening. Some of the patients are confined to bed, some remain in the house, some continue at their work although they feel miserably. The fever lasts a few days, or even two or three weeks.

2. Cases characterized by severe pain in addition to the fever. The pain may be a frontal headache, sometimes of great severity; or the pain may be referred to the back, the limbs, or the thorax. The duration of the disease in these cases is usually only a few days, but it may be protracted for two or three weeks.

3. Cases characterized by great bodily and mental prostration in addition to the fever. In these patients, although the bodily depression is distressing, the mental depression is much worse. It approaches melancholia, and may even pass into it. The fever usually only lasts a few days, but the mental and bodily depression may continue for weeks or months.

4. Cases characterized by acute catarrhal inflammations of the nose, pharynx, tonsils, or larynx, in addition to the fever. In these patients there are the ordinary local symptoms which belong to the inflammations of these mucous membranes. The disease, as a rule, runs its course inside of a week.

5. Cases characterized by bronchitis, which may be developed at any time in the course of the influenza, or even after it has subsided. The bronchitis is of mild type; or severe, with profuse expectoration of mucus with more or less blood; or severe with venous congestion and dyspnoea; or localized in some one part of the lung.

6. Cases characterized by pneumonia. In children the type is that of broncho-pneumonia, in adults that of lobar pneumonia. The inflammation of the lung may be developed at any time in the course of the influenza, or after it has subsided. In adults the pneumonia involves one or more lobes, with complete or incomplete hepatization, often with congestion out of proportion to the extent of hepatization. In nearly all the cases there is marked catarrhal bronchitis of both lungs. The invasion is either like that of an ordinary lobar pneumonia, or more gradual. The physical signs are those of the bronchitis and of the consolidation. The course of the temperature is not like that of an ordinary pneumonia; there is no sudden defervescence, and the fever sometimes continues after resolution has taken place. The expectoration is usually very profuse, more like that of bronchitis than like that of pneumonia. There is in many cases a marked disposition to general venous congestion, with failure of the heart's action. The duration of these pneumonias is often longer than that of ordinary lobar pneumonia, and the resolution slower.

7. Cases characterized by inflammation of the pleura. Dry pleurisies involving a part or the whole of the pleura on one side of the chest are not uncommon. They occur at any time in the course of the influenza. They are accompanied with pleuritic friction sounds, more or less pain, and a moderate rise of temperature. Pleurisies with the production of fibrin and serum, or with fibrin, serum, and pus, are usually not primary, but follow a lobar pneumonia.

8. Cases characterized by acute catarrhal gastritis. The patients suffer from gastric pain, nausea, vomiting, and irritability of the stomach, occasionally with vomiting of blood. These symptoms last only for a few days or are continued for several

weeks. In the protracted cases the emaciation and prostration are extreme, and death may follow.

9. Cases characterized by acute catarrhal colitis. The patients have colicky pains, loose, diarrhoeal passages, a moderate rise of temperature. The inflammation runs its course within two weeks, and the patients are not at any time very sick.

10. Cases characterized by a variety of rashes in the skin.

11. Cases which behave like acute cerebro-spinal meningitis. The patients have a febrile movement which runs an irregular course. They are much prostrated and look very ill. They have the ordinary cerebral symptoms: headache, photophobia, pain, tenderness, and contraction of the muscles of the back of the neck, vomiting, delirium, stupor, general convulsions.

12. The cases with inflammation of the sheaths of the nerves suffer from a good deal of pain along the course of the nerves, followed by loss of power and atrophy of the muscles.

In all these different forms of influenza, after the disease has run its course, the patient may return to his natural health within a short time. But one of the distressing features of the disease in many cases has been the mental and bodily depression, which continues for weeks and months after the acute symptoms have subsided.

*Treatment.*—For the influenza as such there is no treatment.

The headache, if dependent on inflammation of the frontal sinuses, can be relieved in some cases by local applications to the nose. For the general headache and for the pains in other parts of the body, phenacetine, given in five-grain doses every two hours, is the most reliable drug. Of less efficacy for the same purpose are antifebrin, antipyrin, morphine, and the bromides.

The inflammations of the pharynx and tonsils may be alleviated by the local application of cocaine and the internal use of aconite and the salicylate of soda.

For the bronchitis and pneumonia we employ the ordinary methods of treatment, except in the cases with a disposition to general nervous congestion and failure of the heart's action. In these cases attention to the condition of the circulation is of the greatest consequence. I have obtained the best results by the combined use of digitalin  $\frac{1}{8}$  milligm., aconitia  $\frac{1}{6}$  milligm., and whiskey.

For the gastritis we employ small doses of morphine, cocaine in doses of  $\frac{1}{16}$  of a grain, sulphonal in three-grain doses mixed

with the milk. No matter how irritable the stomach may be, I do not believe it is wise to let the patients go for many days without feeding them by the mouth. Feeding by the rectum amounts to very little for these patients.

The colitis is to be treated by rest in bed, a restricted diet, and a little opium.

#### ASIATIC CHOLERA.

An infectious disease characterized by vomiting, purging, cramps, and collapse.

*Causes and History.*—The natural home of the disease is in India, where it has been endemic for an unknown length of time.

The first epidemic of cholera was between the years 1817 and 1823. The disease prevailed all over India, and extended to Ceylon, Burmah, Siam, Japan, China, Arabia, Persia, and Syria.

The second epidemic was between the years 1826 and 1837. It spread through the same countries as in the first epidemic, and extended into Egypt, Russia, Germany, Austria, England, France, Belgium, the Netherlands, Canada, the United States, Mexico, Central America, Portugal, Spain, and Italy.

The third epidemic was from 1846 to 1863, and extended over the same countries as were involved in the second epidemic.

The fourth epidemic, from 1863 to 1875, advanced rapidly into Europe, and was very extensive. In New York the disease prevailed from 1866 to 1867. In the valley of the Mississippi River there was an epidemic in 1873.

The fifth epidemic began in 1883, and is not yet finished. It has as yet not gone over as many countries as did its predecessors.

The disease apparently originates only in India, and does not occur in other countries unless imported into them.

When the disease has been imported into any country it is not likely to spread, unless it finds there conditions favorable to its development. These favorable conditions are: the uncleanly habits of the population, and conditions likely to produce diarrhoea.

The pathogenic micro-organism of the disease was described by Koch in 1884. It is called the "*Spirillum cholerae asiaticæ*," or popularly the "Comma Bacillus."

The micro-organism grows and multiplies rapidly within the intestines, and acts there as a local irritant. It is discharged with the fæces, rarely with the vomit. It is not present in the urine,



the breath, or the sputa, it is not given off from the surface of the body. Outside the human body the organisms live, grow, and multiply on moist surfaces and in dirty water.

They are destroyed by drying, heat, boiling, mineral acids, corrosive sublimate, and carbolic acid.

To communicate the disease the organisms discharged in the fæces of the sick must be taken into the alimentary canals of the healthy.

*Lesions.*—1. Those who die during the stages of invasion or collapse.

The temperature of the body may rise after death. The rigor mortis begins early and lasts for a long time. The skin is of a dusky-gray color; the lips, eyelids, fingers, and toes are livid; the cheeks are fallen in; the skin of the fingers is shrunken and wrinkled.

The sinuses of the dura mater are filled with thick blood. The pia mater may be œdematous, or ecchymotic, or infiltrated with fibrin.

The pleura may be coated with fibrin. The lungs are shrunken and anæmic.

The peritoneum may be coated with fibrin.

The stomach may show the lesions of catarrhal gastritis.

The small intestines are anæmic or congested; the solitary and agminated glands may be swollen; the mucous membrane is soft.

The colon is, in some epidemics, the seat of croupous inflammation.

The kidneys show the lesions of acute degeneration.

2. Those who die during the stage of reaction. The bodies do not present the same appearance of desiccation, and more inflammatory changes are found.

The larynx, bronchi, lungs, pleura, stomach, or intestines may be inflamed.

*Symptoms.*—In the first stage of cholera the disease follows one of two forms:

1. The patients are suddenly attacked with pains in the abdomen, purging, vomiting, and cramps. These symptoms last for half an hour, or several hours. Then the patients either die or go on to the second stage of the disease.

2. The patients have an ordinary, moderate diarrhœa, with loss of appetite, nausea, and prostration. These symptoms con-

tinue for several days. Then the patients recover, or go on to the second stage of the disease.

In the second stage of the disease there are frequent, large passages of fluid, looking like rice-water, from the bowels. There is vomiting of food, of drink, and of a rice-water fluid like that which is passed from the intestines. The patients suffer from constant thirst, from pains in the abdomen, and from painful cramps in the muscles of the legs. The temperature falls below the normal, the pulse is rapid and feeble, the face is shrunken and pinched, the urine is scanty. The second stage lasts for from two to fifteen hours, then the patients may get better, or they may pass into the third stage of the disease.

In the third stage of the disease, the stage of collapse, the vomiting and purging continue, but are not as frequent. The cramps and pains also continue. The heart's action is so feeble and rapid that the radial pulse cannot be felt. The shrunken and livid appearance of the skin and face is more marked; the prostration is extreme; the urine is suppressed; the temperature falls to about  $94^{\circ}$ ; the patients are constantly thirsty, restless and sleepless, but the intelligence remains clear. This stage seldom lasts over twenty-four hours. A great many of the patients die during this stage, a few recover, a few go on to the fourth stage.

In the fourth stage, that of reaction, the skin continues to feel cold, but the temperature rises to  $99^{\circ}$  or  $100^{\circ}$ . The purging and vomiting cease; the radial pulse can again be felt; the breathing is rapid; the urine is suppressed; the patients are semi-comatose. One or other of the inflammations of the pia mater, larynx, lungs, stomach, or intestines is developed, or abscesses are formed in the parotid, or in other places. Very few of the patients who pass into this stage recover.

*Treatment.*—The most important part of the treatment of cholera is its prevention. It is evident, when we remember the way in which the poison of cholera is eliminated from the body, the way in which it is taken into the body, and the ways in which it can be destroyed, that it ought not to be difficult to prevent the spread of the disease. It is only necessary to disinfect the fæces, and to see that infected fæces are not introduced into the mouth with water, food, etc.

During the stage of premonitory diarrhœa the patients should be kept in bed. The bowels should be first emptied and

then full doses of salol, or beta naphthol bismuth, are given, to which a little opium may be added. Or large injections of tannin may be given by the rectum. The formula is :

Infusion of Chamomile Flowers.....	2,000 parts
Ac. Tannic.....	20 parts
Gum-arabic.....	30 parts
Tr. Opii.....	2 parts

Two quarts of the solution at the temperature of 100° F. should be allowed to flow in slowly from a fountain syringe.

During the second stage of the disease the patient can be made more comfortable by hypodermic injections of morphine. The vomiting may be alleviated by cocaine, chloroform, and acidulated drinks. Alcoholic stimulants are usually required. To make up for the loss of blood serum we may use warm baths, or the warm rectal injections of tannin, or subcutaneous injections of warm solutions of chloride of sodium, 80 grammes, carbonate of sodium, 6 grammes, water, 2 litres. This solution is used at the temperature of 110° F., and about thirty ounces can be introduced under the skin at one time.

#### YELLOW FEVER.

*Causes.*—Yellow fever is endemic in the West India Islands, in some places on the shores of the Gulf of Mexico, and on the west coast of Africa.

From these places the disease has been carried to different places in the United States, Central America, South America, and Spain, producing epidemics in these places.

We are still ignorant of the micro-organism which probably constitutes the poison of the disease ; nor do we know how it is eliminated from, or taken into, the body.

The poison of the disease requires a temperature of 70° F. for its growth and development. It is capable of remaining in clothing, bedding, ships, etc., and retaining its vitality for a long time.

Cities are more liable to the disease than country places ; dirty cities are more liable than clean ones.

The inhabitants of countries where the disease is endemic are less liable to it than are strangers. One attack confers a less liability to a second attack.

*Lesions.*—There is well-marked jaundice of the skin and other tissues. There are often ecchymoses in the skin.

The wall of the heart is in the condition of granular and fatty degeneration.

The lungs are congested.

The stomach contains more or less black fluid, its mucous membrane is congested. The intestines may contain blood.

The liver shows the changes of acute degeneration. The liver-cells are swollen, coarsely granular, or disintegrated.

In the kidneys we find the changes belonging to an intense form of acute degeneration. The kidneys are large, the renal epithelium is degenerated, or dead, the tubes contain cast matter.

*The period of incubation* of yellow fever varies from twenty-four hours to several weeks ; usually it only lasts for a few days.

*Symptoms.*—Occasionally there is a prodromic period, lasting for several days, marked by headache, giddiness, and loss of appetite, but more frequently the invasion is sudden, marked with chill, or sometimes with general convulsions.

During the first stage the temperature runs up rapidly, reaching 103° F. to 110° F. by the end of the first twenty-four hours. The pulse is a full pulse of 80 to 120, but sometimes is very slow. There are headache, pains in the back, restlessness, sleeplessness, delirium, vomiting of food, constipation. The face is flushed, the tongue is coated, the urine contains albumin and casts.

Exceptionally, during the first stage the fever is moderate, there is no delirium, but little prostration, and the patients are not confined to bed.

The first stage lasts for from two to six days.

During the second stage of the disease the temperature and the pulse fall to the normal, and the patients are better in every way. Some then go on to convalescence ; others, after one or two days, go on to the third stage.

During the third stage the vomiting of food continues, and is succeeded by the vomiting of a blackish fluid, the so-called black vomit. The patients become jaundiced, are dull and stupid, and complain of pain in the abdomen. There may be bleeding from the nose, mouth, intestines, and kidneys, or into the skin and connective tissue.

The urine is very scanty or suppressed.

The conditions of alternating stupor and delirium are very marked.

The third stage lasts for from one to three days.

The mortality of the disease is very high.

*Treatment.*—The preventive treatment consists in isolating the patients, and in destroying or disinfecting the clothes, bedding, etc.

The treatment consists principally in an attempt to alleviate symptoms.

The bichloride of mercury, combined with soda, has been recommended for the treatment of the disease itself.

Cocaine, given by the mouth, is used as a remedy for the vomiting.

#### TYPHUS FEVER.

*Synonyms.*—Typhus exanthematicus. Putrid, camp, jail, hospital, ship, or spotted fever.

*History and Causes.*—Typhus fever is endemic in Ireland, England, and Russia. In other countries it occurs from time to time in epidemics. It has regularly accompanied wars, famine, and the crowding of prisons, ships, and houses with ill nourished and ill-aerated human beings.

Epidemics have occurred in this way in all parts of Europe.

In the United States there have been a few epidemics, mostly in the cities on the sea-coast.

In New York the last severe epidemic was in 1863 to 1864. In 1882 there were a few cases of typhus in the city, but hardly an epidemic.

Although no age is exempt from the disease, the maximum liability is between the ages of fifteen and twenty years.

The disease is apparently not influenced by the seasons of the year or by atmospheric conditions.

The poison of the disease has not as yet been demonstrated. It seems to be given off from the surface of the sick body, to float in the air, and to contaminate bedding, clothing, etc. So we find that the disease is communicated from person to person, and by clothing, bedding, and rooms. Nurses and physicians very often contract the disease. It is much more easily taken by persons who are poor, badly fed, and over-crowded. Few people escape the disease if they are sufficiently exposed. It seems to be most contagious from the end of the first week up to the time of convalescence.

The accumulation of patients in the same room renders the disease more contagious.

It is an exceptional thing for any person to suffer from more than one attack of the disease.

*Period of Incubation.*—The symptoms may appear a few hours after exposure. Usually they appear between eight and twelve days after exposure ; rarely, if ever, after a longer period than twenty-one days.

*Lesions.*—The only characteristic lesion of the disease is the eruption. But after death a number of secondary lesions may be found.

There may be acute meningitis. There may be catarrhal or croupous inflammation of the pharynx and larynx.

In the lungs we find bronchitis, broncho-pneumonia, hypostatic congestion, or œdema.

In the heart there may be degeneration of the muscular fibres.

The liver and spleen may be the seat of acute degeneration.

In the ileum the agminated glands may be a little swollen, as may also the mesenteric glands.

The voluntary muscles in different parts of the body may be the seat of acute degeneration.

In the kidneys we find the lesions of the mild or severe forms of acute degeneration, or those of acute exudative nephritis.

*Symptoms.*—During the first week there is a general venous congestion of the face and conjunctiva, and a dull, apathetic expression. During the second week the patients look like persons in the third week of typhoid fever.

The tongue is at first coated and moist, later dry and brown.

Cerebral symptoms are marked—restlessness, sleeplessness, delirium, and stupor. The delirium is attended with illusions, is often violent, and is worse at night. It may mark the invasion of the disease, it may simulate an attack of acute mania, or it may not be developed until the second week. It is always a dangerous delirium, and one which needs constant watching. As the delirium subsides the stupor increases, but without natural sleep.

Sometimes the patients develop the condition called “coma vigil.” They are unconscious, with open eyes, a rapid and feeble pulse, shallow breathing, and a cold skin.

Headache and pains in the back and limbs are early and frequent symptoms.

Vomiting and tympanites are not very common.

Constipation is the rule until the close of the disease, but there may be diarrhoea throughout.

The prostration is marked, but during the first week may be less evident by reason of the active delirium.

The eruption is very constant, but varies in its extent. It appears from the fourth to the seventh day, and lasts from seven to ten days. There is only a single crop. It appears on the arms, legs, and trunk. It is in the form of irregular, rounded, pinkish blotches resembling the eruption of measles. The spots become darker with time, or may be quite dark from the first. There is also sometimes a deep mottling in the skin, and sometimes a general erythematous blush. From the eighth to the tenth day there may be small ecchymoses in the blotches of the eruption. After the eruption has disappeared there is more or less desquamation.

The temperature rises rapidly, and reaches its maximum between the second and tenth day, usually between the fourth and seventh day, reaching  $104^{\circ}$  to  $106^{\circ}$  F. At first the temperature is nearly continuous, by the seventh to the tenth day there are morning remissions. In favorable cases, about the fourteenth day the temperature suddenly falls.

The pulse is at first about 100 and full, later it becomes rapid and feeble. Or it may be rapid and feeble from the first, or slow and feeble throughout the disease.

The heart's action in the second week becomes feeble, and its first sound less distinct.

The breathing is rapid, and in the second week is rendered worse by the hypostatic congestion, œdema, bronchitis, or broncho-pneumonia.

The kidneys become the seat of acute degeneration, or acute exudative nephritis, with diminution in the quantity of urine, and more or less albumin and casts.

Muscular tremor, twitching of the muscles of the face, sub-sultus tendinum, picking at the bed-clothes, and automatic movements of the arms and legs may be developed during the second week.

General convulsions sometimes occur in the second week, with the severer forms of nephritis.

In the later stages of the disease there is retention of urine, incontinence of fæces, and difficulty in swallowing.

Severe catarrhal or croupous inflammations of the pharynx or larynx are sometimes developed.

Acute meningitis, with an exaggeration of the cerebral symptoms belonging to the typhus fever is a dangerous complication.

There may be bleeding from the mucous membranes into the skin, the serous membranes, and the muscles. The most dangerous hemorrhages are those from the stomach.

Often there is suppuration of one or both parotid glands.

Not infrequently there is thrombosis of one or both femoral veins, less frequently of the sinuses of the dura mater.

Bed-sores are easily formed and very troublesome.

*Course of the Disease.*—There may be a prodromic period marked by general malaise, headache, vertigo, and loss of appetite.

The invasion of the disease is usually sudden, marked by chills, fever, headache, pains in the back and limbs, loss of appetite, vomiting, restlessness, sleeplessness, and prostration. From the fourth to the seventh day the eruption appears.

In the second week come the alternating delirium and stupor, the dry tongue, the typhoid state, the feeble heart-action, the complicating lesions.

In the favorable cases, about the fourteenth day the temperature falls, the patients begin to sleep, and then convalescence commences.

The regular duration of the disease is fourteen days. Murchison, in 500 cases of recovery, gives the subsidence of the disease as between the seventh and the twentieth days; in three-fourths of the cases between the thirteenth and sixteenth days. In 100 fatal cases death took place between the sixth and the twentieth days. There are some very bad cases which only live a few hours.

*Sequelæ.*—In some cases a condition of mental imbecility continues for several weeks.

In some patients aphasia or hemiplegia follow the disease.

There may be paralysis of groups of the voluntary muscles.

*The prognosis* is always grave. The disease is especially fatal in elderly persons and in those enfeebled by starvation. Some epidemics are more fatal than others.

*Treatment.*—The patients are to be isolated as far as possible. It is important not to accumulate many patients in the same room or building, and to provide for the largest possible quantity of fresh air. The clothing and bedding must be burned or thoroughly disinfected.



In the treatment of the disease the most important points are the very large admission of fresh air and the nursing. Alcoholic stimulants in large quantities are often of service, but must be given with judgment.

#### TYPHOID FEVER.

An infectious disease characterized by fever and the development of lesions in the lymphatic glands of the intestine and mesentery.

*Lesions.*—1. The characteristic lesions :

By the second day of the disease the solitary and agminated glands of the intestine are swollen, and the swelling increases during the first week. In the second week there is more or less necrosis of the swollen glands, with ulceration or sloughing. In the fourth week there is cicatrization of the ulcers.

The swelling of the glands may be moderate and not go on to ulceration or sloughing ; it may be greater with the formation of small ulcers ; it may be very great with extensive necrosis and sloughing.

A number of the glands are attacked at the same time, or first those in the lower part of the ileum are involved, and later those farther up.

The lesions may be confined to the agminated glands, or involve also the solitary glands, or extend to the solitary glands of the colon.

The cases vary as to the number of glands involved, the degree of the swelling, the size and depth of the ulcers.

The mesenteric glands are usually only swollen, sometimes they suppurate. They may excite a local or a general peritonitis.

2. The accessory lesions :

The spleen is usually large and soft. In the ileum and colon there may be catarrhal or croupous inflammation.

There may be peritonitis from perforation of the intestinal ulcers, from the diseased mesenteric glands, or without discoverable cause.

The softened spleen may rupture. The hepatic cells may be the seat of acute degeneration.

The kidneys exhibit the lesions of acute degeneration, or of acute exudative nephritis.

The heart-walls are often soft and flabby. The parotid glands

may suppurate. In the pharynx and larynx there may be catarrhal or croupous inflammation.

The lungs are often the seat of hypostatic congestion, of bronchitis, of broncho-pneumonia, or of lobar pneumonia.

There may be acute meningitis. The peripheral nerves may be the seat of an acute degeneration.

There is degeneration, and sometimes rupture, of the voluntary muscles.

There may be thrombosis of the femoral veins, or of the sinuses of the dura mater.

The characteristic micro-organism of the disease is a bacillus which is found in the intestinal lesions and in the contents of the intestines. It is also found in the mesenteric glands, the spleen, the liver, the kidneys, and the blood.

The bacilli are discharged from the typhoid patient principally with the fæces. They can live for a long time outside of the body, under favorable conditions.

They are most frequently taken into the body with contaminated water and milk.

The course of the growth of the bacilli seems to be as follows: They are taken into the stomach, pass into the small intestine, and remain there without giving any symptoms for about two weeks. Then they begin to find their way into the lymphatic glands in the wall of the small intestine. As soon as this happens the patients have symptoms more or less severe, according to the extent of the infection of the glands. At a later time the bacilli find their way to the mesenteric glands, the spleen, and other parts of the body. The effect of the presence of the bacilli on the lymphatic glands in the intestine is to produce inflammation, or necrosis, or both. The growth of the bacilli is attended with the evolution of a poison which is the direct cause of the symptoms of the disease.

*Etiology.*—Typhoid fever prevails in the form of local epidemics, as an endemic disease, and in the form of isolated cases.

The history of a local epidemic is apt to be as follows: A person already suffering from typhoid fever goes to a house in the country, or in a village where the disease has not previously existed. He goes through the disease at this place. His fæces are put in a privy vault, or cesspool; his soiled clothes and bedding are washed without being disinfected. The persons who handle and wash the soiled clothes often contract the disease. The contents

of the infected privy or cesspool after a time drain into the adjoining well ; most of the persons who drink this contaminated water have typhoid fever. The persons living in the same village who do not drink the contaminated water, nor handle the infected clothes, do not have the disease.

Occasionally the disease is given by milk which has been contaminated by the infected water.

In some places typhoid fever is an endemic disease, prevailing year after year. When this is the case the disease is regularly much more prevalent in the autumn months, and varies in different years as to the number of cases and the type of the disease.

The most puzzling cases to account for are the isolated cases which occur from time to time in cities.

The disease seems to protect against itself, and the same person rarely suffers from two attacks.

*Symptoms.*—The regular duration of the disease is four weeks, and to each week belongs its own set of symptoms.

The regular course of the temperature is as follows :

During the first week the temperature rises regularly from day to day, always with a lower temperature in the morning and a higher temperature in the evening, but with each day the difference between the morning and evening temperature becomes less.

During the second week the temperature is always high, with but little difference between the morning and evening temperature.

In the third week the morning temperature begins to fall, the evening temperature remaining the same.

In the fourth week the morning temperature falls still lower, then the evening temperature falls, until finally both get down to the normal.

The maximum temperature is reached between 5 and 12 P.M. The minimum temperature is recorded between 6 and 8 A.M.

*Variations from the Regular Temperature.*—In the first week the invasion may be ushered in by a chill, and similar chills may be repeated for several days. The temperature may fall to the normal for as long as forty-eight hours.

The temperature may rise rapidly and reach 104° F. by the first, second, or third day.

In the second week there may be no difference between the

morning and evening temperature. There may be an unusual difference between the morning and evening temperature. The morning temperature may be the highest. There may be irregular rises and falls of temperature from day to day.

In the third week the temperature may be higher than at any time in the disease. The temperature may suddenly fall to the normal and not rise again, or it may fall and then go on again. There may be irregular changes in the temperature from day to day.

In the fourth week the temperature may fall to the normal, then rise and go on for from five to ten weeks.

In the third and fourth weeks the morning temperature may fall to the normal or below it, while the evening temperature runs up to  $105^{\circ}$  or  $106^{\circ}$  F. This makes one think of a complication by malarial poisoning. But it may occur with nothing but the typhoid fever.

Complicating inflammations change the course of the temperature.

Hemorrhages from the bowels are often followed by a marked fall of temperature.

An evening fever, sometimes followed by sweating, may be continued after the fourth week.

In fatal cases the temperature usually continues high up to death. But sometimes the temperature is not higher than  $100^{\circ}$  to  $102^{\circ}$  F. for several days before death.

The height of the temperature is regularly in proportion to the severity of the disease, but in some fatal cases the temperature is never very high.

The course of the temperature is changed by malarial poisoning.

If the patient is regularly bathed the course of the temperature is very much modified.

The pulse regularly follows the temperature. During the first week it is from 80 to 100 and full; during the second and third weeks it is more rapid, more feeble, or dicrotic.

The feeble pulses are usually rapid, but sometimes slow.

The disease may run its entire course with a pulse between 60 and 80.

*Cerebral Symptoms.*—Headache, restlessness, irritability, and sleeplessness belong to the first week of the disease.

In the second week the patients are in a condition of apathy.

In the third week are regularly developed alternating delirium and stupor.

A violent delirium may come on in the first days of the disease.

The characteristic apathy may be absent, even in fatal cases.

There are some patients in whom the headache is very severe at first, and in whom there is later developed a stupor which continues until death. These cases resemble cerebro-spinal meningitis.

The eruption is in the form of small, isolated, rose-colored, lenticular spots, slightly elevated, and disappearing on pressure. It appears on the front of the chest and abdomen, sometimes on the back. There are only a few spots, or many. It appears between the seventh and twelfth days, exceptionally between the fourth and twentieth days. There are successive crops of eruption, so that it lasts from seven to twenty-one days, but it may only last for from two to four days.

Relapses are marked by a fresh eruption. Spots may continue to appear after convalescence has commenced.

In some cases no eruption can be demonstrated.

The tongue is at first moist, with a broad strip of white fur down the centre. In the milder cases the tongue remains moist throughout the disease, in the more severe cases it becomes dry, brown, and fissured. Or the tongue remains clean, but becomes dry, glazed, and fissured.

Nausea and vomiting are often present during the first week. Later in the disease there may be vomiting from the tympanites, from the retention of food in the stomach, or from peritonitis.

Some distention of the intestines with gas is usually present; marked distention is an unfavorable symptom; extreme distention may so interfere with the action of the heart and lungs as to cause alarming pulmonary and cardiac symptoms.

Diarrhœa is a regular symptom of the disease, several loose, fecal passages every day, of the appearance of pea-soup. Such a diarrhœa may be one of the first symptoms; it may not come on until the third or fourth week; it may not begin until after the patient has taken a purgative; it may last for a few days or throughout the disease. A severe diarrhœa is an unfavorable symptom. In some epidemics but few patients have any diarrhœa at all.

Hemorrhage from the intestines occurs in a moderate num-

ber of the cases. During the first week there may be moderate bleeding from the congested mucous membrane.

The more important bleedings are from the ulcers in the intestines. They occur most frequently in the third and fourth weeks, but are sometimes seen as early as the second week, sometimes not until after the fourth week, or even in convalescence. The hemorrhages are small or large, single or repeated. Large hemorrhages are regularly followed by a temporary fall of temperature. The hemorrhages are usually, but not always, preceded by diarrhœa. Such hemorrhages are sometimes fatal, and it is even possible for a patient to bleed to death in this way without the escape of any blood from the rectum.

There is often epistaxis, either early or late in the disease, single or repeated, rarely so profuse as to be dangerous.

The eyes in the third week may become somewhat insensible to light, with dilated pupils.

Ringings and buzzing sounds in the ears may be complained of in the first week; in the third and fourth weeks there may be more or less deafness.

Subsultus tendinum, muscular tremor, picking at the bed-clothes, the involuntary passage of fæces, and retention of urine belong to the third and fourth weeks.

The condition of the urine varies with that of the kidneys. The most common lesion is a mild form of acute degeneration, with a little albumin and a few casts in the urine. In the protracted cases, however, the degeneration of the epithelium becomes very marked. Less frequently there is an acute exudative nephritis, with diminution in the quantity of urine, numerous casts, and considerable quantities of albumin.

Bronchitis may be developed at any time in the disease. It is usually confined to the larger tubes, and it is not very severe.

Broncho-pneumonia is a much more serious complication. It may be developed at any time in the course of the disease. When it occurs in the first week, it is often very difficult to tell whether the patients have pneumonia or typhoid fever complicated by pneumonia.

Catarrhal pharyngitis is very often present, croupous pharyngitis but seldom.

Catarrhal or croupous laryngitis, ulcers of the larynx, and œdema of the glottis are occasionally met with.

Suppurative inflammation of the parotid glands belongs to the later periods of the disease.

During the third and fourth weeks, sometimes earlier, there may be thrombosis of the veins. The veins most frequently affected are the femoral veins and the sinuses of the dura mater. The formation of a thrombus is attended with a rise of temperature and increased prostration. With thrombosis of the femoral vein there is pain and tenderness over the vein and œdema of the leg.

With thrombosis of the sinuses of the dura mater there is loss of consciousness or hemiplegia, according to the position of the thrombus.

From thrombosis of the femoral vein patients usually recover, but thrombosis of the sinuses of the dura mater is usually fatal.

In some patients the disposition to bleeding from the different mucous membranes is very marked.

Catarrhal or croupous inflammation of the colon may accompany or follow typhoid fever.

Perforation of the intestinal ulcers is a very fatal lesion. It happens most frequently in the third, fourth, and fifth weeks. It may happen in the second week, or after convalescence is established. It is most common in severe cases, but may occur in the very mild ones. It is followed by the symptoms of shock and the development of a general peritonitis.

Peritonitis is produced not only by perforation, but also by an extension of the inflammation from the wall of the intestines, by inflammation of the mesenteric glands, and by infarctions of the spleen. It usually gives well-marked symptoms of acute general peritonitis, and is very fatal.

*Course of the Disease.*—The period of incubation is said to vary between two days and four weeks; the ordinary period is two weeks.

The method of invasion of the symptoms varies considerably in different cases. This difference seems to depend upon differences in the growth of the typhoid bacilli and the rapidity with which they find their way into the wall of the intestine, the spleen, and the rest of the body.

In a considerable number of cases one or two weeks elapse before the patients feel sick enough to go to bed. They are more or less miserable, with headache, sleeplessness, nausea, an irregular febrile movement, and sometimes diarrhœa. This seems

to mean that the bacilli are growing in the intestine, are beginning to find their way into its wall, but that the quantity of poison evolved by their growth is but small.

The more ordinary invasion is for the patients to feel sick enough to go to bed within the first twenty-four hours. Then for about two weeks they get steadily worse. The temperature becomes gradually higher and more continuous, the patients suffer from epistaxis, pharyngitis, headache, vertigo, apathy, sleeplessness, pain in the neck, back, and abdomen, a coated tongue, loss of appetite, nausea, vomiting, diarrhœa, or constipation. This would correspond with a sudden and extensive invasion of the lymphatic glands of the intestine, a gradual but continuous invasion of other parts of the body, with a very considerable evolution of poison.

Occasionally it happens that while the patient becomes quite ill, with a rise of temperature, during the first twenty-four hours, yet for the ten succeeding days there are intermissions of low temperature and subsidence of symptoms. After this the patient settles down into the regular course of the disease. This would correspond with interruptions in the growth and invasion of the bacilli.

It may also happen that the patient begins with the regular invasion of a mild typhoid and goes on to be moderately sick for two weeks. At the end of that time the symptoms change for the worse and the disease runs a severe course. This would seem to indicate a second infection more severe than the first.

The invasion of the symptoms may be unusually rapid and severe. The temperature runs up to 104° F., with chills, within the first twenty-four hours. Here the infection is evidently very rapid and extensive.

There are irregular invasions for which we are unable to account, and which simulate the invasions of other diseases.

In some patients the first symptoms resemble those of cerebro-spinal meningitis: headache, pain, and stiffness in the back of the neck, violent delirium or general convulsions.

In some patients the complicating inflammations of the chest are very marked. It may be a bronchitis, or a broncho-pneumonia, or a pleurisy.

Or instead of this there is an acute catarrhal gastritis with vomiting and diarrhœa; or an acute nephritis with the characteristic changes in the urine.



During the first week there are the characteristic temperature, epistaxis, pharyngitis, headache, vertigo, apathy, sleeplessness, pain in the neck, back, and abdomen, the coated tongue, loss of appetite, nausea, vomiting, diarrhœa, or constipation, blood with the stools, prostration.

It is often difficult to fix the date of the first day of the disease. The ordinary rule is to take the first day when the patient takes to his bed.

During the second week of the disease there are the characteristic temperature, the increased rapidity and feebleness of the heart's action, more apathy, delirium, noises in the ears, deafness, the dry tongue, the eruption, diarrhœa or constipation, tympanites.

At the end of the second week the patient may die, he may become convalescent, or the disease may continue.

During the third week there are the characteristic temperature, the heart's action more rapid and feeble, the cerebral symptoms more marked, the full development of the typhoid state, bleeding from the intestinal ulcers, peritonitis, involuntary passage of fæces, retention of urine.

In the fourth week, if the case is a favorable one, there is a gradual abatement of all the symptoms, and convalescence commences in the fifth week. If the disease is to be protracted, the symptoms of the fourth week will continue like those of the third, or there will be an improvement for one or two days, and then the disease goes on again.

The relapses of typhoid fever are a curious feature of the disease. They seem to be a fresh attack of the disease produced by a fresh infection from the patient himself. Each relapse is attended with a new eruption and a repetition of the symptoms of the disease.

The intermission between the end of the original attack and the beginning of the relapse is from three to twenty-five days, the ordinary period is from ten to fourteen days. The relapse lasts from seven to thirty-nine days, the ordinary duration is from ten to fourteen days. A single relapse is not uncommon, more common in some epidemics than in others. A second relapse is comparatively rare.

*The Irregular Cases.*—1. The Mild Form. The pulse and temperature remain nearly or quite normal. The patients have irregular feelings of heat and cold, headache, irritability, sleep-

lessness, loss of appetite, and general prostration and discomfort, but are not sick enough to be in bed. The bowels continue regular, or there may be constipation or diarrhœa.

In some of the patients bronchitis is a prominent symptom, in others nausea and vomiting.

The patients remain in this condition for three or four weeks. They are liable to hemorrhage from the intestine, to perforation of the intestinal ulcers, and to attacks of delirium.

2. The Short Form. The whole duration of the disease is only two weeks.

In some of the patients there is a considerable rise of temperature, of which the rise and fall are alike rapid.

In other cases there is no fever, only lassitude, headache, and loss of appetite.

3. The Protracted Form. Instead of running its course within four weeks the disease continues. The fever and other symptoms go on with little change, and the patient remains ill for five, six, seven, eight, nine, ten, eleven, or even twelve weeks, as the case may be.

4. The Afebrile Form. Typhoid fever may run its entire course with evening temperatures of not over  $99^{\circ}$ , and morning temperatures below  $98^{\circ}$ , and yet it is evident that the patients are really ill. They often feel exceedingly weak, with a feeble heart-action.

In 1870 there was an epidemic of typhoid fever in the German army around Paris. The cases were characterized by an abundant eruption, great prostration, violent delirium, marked stupor, but slight abdominal symptoms, and temperatures not over  $100^{\circ}$ , or not above  $98^{\circ}$ . The disease was moderately fatal, and after death the ordinary lesions were found.

5. Typhoid Fever Complicated by Malarial Poisoning. It is possible for a patient to be infected at the same time by the poisons of typhoid fever and malaria. He will then go through a disease with lesions and symptoms belonging to both these morbid states.

*Convalescence* begins regularly in the fifth week. If uncomplicated it progresses steadily, but yet it is months before the patient is really like himself.

The convalescence may be interrupted in several ways :

An afternoon fever may continue for a number of days.

There may be severe pain in the back, apparently due to inflammation of the periosteum of the vertebræ.

The mind may remain dull, apathetic, and feeble for days or weeks.

There may be hemiplegia, dependent on thrombosis of the sinuses of the dura mater; or paralysees of groups of muscles due to changes in the peripheral nerves.

There may be hemorrhage from intestinal ulcers which have not healed, or perforation with peritonitis.

There may be a catarrhal gastritis, with pain, nausea, and vomiting.

There may be catarrhal or croupous inflammation of the colon, with the symptoms of dysentery.

There may be rapid and irregular heart-action.

The patients, after beginning to convalesce, may make no real progress and die in an emaciated and feeble condition.

*The Prognosis* of typhoid fever is a very uncertain one.

Cases which are at first mild may later turn out very badly.

The cases with high temperatures and with nearly continuous temperatures are always severe.

A profuse and continued diarrhœa is a serious symptom.

Very marked distention of the intestines with gas belongs to the bad cases.

Hemorrhages from intestinal ulcers, even if they are not of themselves fatal, belong to the severe cases of the disease.

Perforation of an intestinal ulcer, or peritonitis from any cause, is nearly uniformly fatal.

Severe muscular tremor indicates the existence of deep intestinal ulcers.

Epistaxis is not often of itself serious, but its early and repeated occurrence belongs to the more severe cases.

The sooner the patient goes to bed after the invasion of the disease, the better the prognosis.

The mortality varies in different epidemics, and in different years, in places where the disease is endemic.

*Treatment.*—1. To prevent the disease.

The fæces should be received into a vessel containing a sufficient quantity of disinfecting fluid, and thoroughly mixed with this fluid before being emptied into the common receptacle.

The following are efficient disinfecting fluids:

R.	Bichloride of mercury.....	3 ij.
	Acid hydrochloric.....	3 x.
	Water.....	1 gallon.
M.		

R.	Bichloride of mercury .....	3 ij.
	Permanganate of potash.....	3 ij.
	Water.....	1 gallon.
M.		
R.	Chloride of lime.....	3 iv.
	Water.....	1 gallon.
M.		

All the soiled clothes are to be soaked in one of these solutions, or steamed before they are washed.

Persons travelling or living in localities where typhoid fever exists must be very careful about drinking the water of the place.

*Treatment of the Disease.*—The patients are to be put to bed and placed on a fluid diet as soon as possible.

During the first week the patients are troubled by headache, restlessness, irritability, and pains in the stomach. These symptoms can be alleviated by sulphonal, opium, and the bromides, but these drugs are to be used sparingly and with caution.

Vomiting can usually be relieved by attention to the food.

If the bowels are constipated, they should be moved once in three days by an enema; if there is too much diarrhœa, it must be checked to some extent by opium. Cold bathing often stops the diarrhœa.

The milder nose-bleeds require no treatment; for the more severe ones it is necessary to plug the nostrils.

If the pharyngitis is troublesome, local applications of cocaine give relief.

The mouth and tongue are to be kept clean.

The heart's action during the first week usually continues good.

As regards the temperature, the following propositions may be stated:

The temperature as such does not add to the dangers of the disease, nor require treatment.

The systematic use of cold baths of the temperature of 65° F. lowers the mortality of the disease, but not because it lowers the temperature. High temperatures may make the patients uncomfortable, and it may, therefore, be proper to give antipyretic drugs, although these drugs do not increase the probability of recovery.

In the second week the heart's action often begins to fail and calls for the use of alcoholic stimulants.

If there is excessive distention of the intestines by gas, we

employ hot or cold applications over the abdomen, the internal administration of turpentine or asafœtida, placing a soft-rubber tube in the rectum, or cold bathing.

For the bleeding from the intestinal ulcers, we give opium and apply cold over the abdomen.

For excessive muscular tremor we give opium, asafœtida, or the compound spirits of ether.

In the third week we have to care for the retention of urine, the formation of bed-sores, the suppurative inflammation of the parotid glands, the thrombosis of the veins, and the peritonitis.

For the disease itself the most efficient treatment is by cold bathing. In carrying out this treatment the best results are obtained by adhering closely to the method as laid down by Brand. Once every three hours, if the temperature in the rectum reaches 103° F., the patient is to be immersed in a bath of the temperature of 70° F. In this bath he is to be kept for ten or fifteen minutes, during which time the surface of the entire body is constantly rubbed. After the bath the patient takes either an alcoholic stimulant or a hot drink. The best results are obtained when the bathing is begun during the first week of the disease.

When the bathing cannot be carried out, the treatment by intestinal antiseptics may be tried.

The method of Burney Yeo is as follows: In a 12-ounce bottle 30 grains of chlorate of potash and 40 minims of hydrochloric acid are mixed. After the bottle is filled with gas it is filled up with water. To 12 ounces of this solution, 24 grains of quinine and 1 ounce of syrup of oranges are added. The dose of this is 1 ounce at intervals of from one to four hours.

Another method is to give sulphate of magnesia in repeated doses so as to produce moderate purgation, the patient at the same time drinking large quantities of water.

Still another plan is that of giving considerable doses of salol, or of carbolic acid.

The plan of Dr. Woodbridge is as follows: The treatment is begun with tablets each of which contains

Podophyllum resin.....	$\frac{1}{960}$ grain.
Calomel.....	$\frac{1}{16}$ grain.
Guaiacol carbonate.....	$\frac{1}{16}$ grain.
Menthol.....	$\frac{1}{16}$ grain.
Eucalyptol.....	q. s.

One tablet is given every 15 minutes during the first 24 hours, and in larger doses if necessary during the second 24 hours, until during this and the succeeding 24 hours not less than five or six free evacuations of the bowels are secured during each of these periods. On the third or fourth day of treatment the following tablets are used :

Podophyllum resin.....	$\frac{1}{960}$ grain.
Calomel.....	$\frac{1}{16}$ grain.
Guaiacol carbonate.....	$\frac{1}{4}$ grain.
Menthol.....	$\frac{1}{16}$ grain.
Thymol.....	$\frac{1}{16}$ grain.
Eucalyptol.....	q. s.

A tablet is given at intervals of one or two hours. Both these tablets are given at longer intervals as the temperature falls.

About the fourth or fifth day of treatment the employment of the following capsules is commenced :

Guaiacol carbonate.....	3 grains.
Thymol.....	1 grain.
Menthol.....	$\frac{1}{2}$ grain.
Eucalyptol.....	5 minims.

One capsule every three hours, alternating with the tablets.

*Convalescence.*—The principal point in a normal convalescence is to manage the transition from fluid to solid food. In my opinion this transition should usually not be delayed for more than a few days, and the best solid food to begin with is beef or mutton.

When the typhoid fever seems to have run its course, and yet an afternoon fever continues, there may be an advantage in the use of quinine, and in these patients we do not always wait for a normal temperature before we begin the use of solid food.

#### RELAPSING FEVER.

*Synonyms.*—Famine fever. Spirillum fever. Typhus recurrens.

*History and Causes.*—Epidemics of this disease have occurred since 1739, in Ireland, England, and Scotland. From 1863 to 1873 there were epidemics in Russia and Germany. In India there were epidemics from 1877 to 1880. In New York there were epidemics in 1847, and in 1869 to 1870.

The disease occurs regularly in epidemics. It has often fol-

lowed famines, and is especially prevalent among the poor and destitute. It is very contagious, but we do not know the exact way in which the disease is transmitted from one person to another. One attack does not protect against subsequent attacks. As a rule, epidemics, when they have run their course, disappear completely, but in a few places the disease has become endemic.

*Lesions.*—The pharynx may be the seat of catarrhal or croupous inflammation.

The stomach may be congested, or there may be extravasations of blood in its mucous membrane.

The small and large intestine may be the seat of catarrhal or croupous inflammation.

The liver may be the seat of acute degeneration and increased in size.

The spleen is large and soft ; it may contain infarctions ; it may rupture.

There may be a general peritonitis. The muscular fibres of the wall of the heart may be degenerated.

The larynx may be the seat of catarrhal or of croupous inflammation.

Bronchitis, broncho-pneumonia, lobar pneumonia, and pleurisy, are not infrequent.

In the kidneys acute degeneration of mild, or of severe, type is regularly present.

The characteristic micro-organism of the disease is a spirillum which is found in large numbers in the blood. It is found most constantly in the febrile stage of the disease. The blood containing the spirilla, when inoculated in monkeys, reproduces the disease. It is, however, not known how in the human subject the micro-organism is eliminated from the body of the sick person, or how it is transferred from one person to another.

The period of incubation of the disease seems to vary from a few hours to twenty-one days.

*Symptoms.*—The invasion of the disease is regularly sudden. There are chills, headache, pains in the back and limbs, and prostration. Very soon the temperature rises, and soon runs up to from 104° to 108° F. The headache is worse, the tongue is coated, there is vomiting of food, of greenish or of coffee-ground matters.

The patients cannot sleep, there are pain, tenderness, and enlargement of the liver and spleen.

Between the third and tenth day, usually on the fifth or seventh, there is an abrupt fall of temperature, with sweating, epistaxis, diarrhœa, or hemorrhages from the bowels or stomach. For about seven days the cessation of fever continues, and then there is a relapse, with all the symptoms of the first attack. The relapse lasts for from one to seven days, usually for three. There may be a second, third, or fourth relapse. Rarely, there is no relapse at all.

There is no characteristic eruption, but in some epidemics roseola, or a reddish mottling of the skin, or petechial spots are present.

The bowels are sometimes constipated, sometimes loose.

Jaundice may appear in the first paroxysm, in the second, or in both. It belongs to the severe cases.

The urine varies with the severity of the acute degeneration of the kidneys. There may be a good deal of pain in the muscles and joints during the disease, or in convalescence.

Rarely, there are general convulsions at the time of the fall of temperature.

*Complications and Sequelæ.*—There may be laryngitis, bronchitis, pleurisy, lobar pneumonia, rupture of the spleen, acute colitis, or peritonitis.

During convalescence there may be inflammation of the ciliary body or choroid coat of the eye; or paralysis of groups of muscles.

*Prognosis.*—The mortality varies with the epidemics and with the previous condition of the patients.

The bad cases are characterized by hemorrhages from the stomach and bowels, cerebral symptoms, suppression of urine, pneumonia, peritonitis, dysentery, and collapse at the time of the fall of temperature.

*Treatment.*—The prophylactic treatment is difficult on account of our ignorance of the way in which the poison of the disease is eliminated from the body.

Of the disease itself, the treatment consists wholly in the nursing and the alleviation of symptoms.

#### THE EXANTHEMATA.

The members of this group of the infectious diseases have several features in common :



Each one of them has a characteristic form of inflammation of the skin.

Each has its own specific poison, which seems to be given off from the skin and inhaled with the breath.

They do not originate from outside causes, but each new case is derived from a preceding case of the same disease.

Each of them has a regular period of incubation, of invasion, and of eruption.

In each of them one attack protects more or less completely against subsequent attacks of the same disease.

#### SMALL-POX.

Small-pox, or variola, seems to be one of the oldest diseases known to us. There are accounts of the disease going back to the sixth century; and from the Middle Ages to the time of the introduction of vaccination the disease, both in its epidemic and endemic forms, was very prevalent and terribly fatal.

*Etiology.*—Although small-pox possesses an exceedingly active poison, we are still ignorant of its exact nature. It is probable that it is a micro-organism, it is certain that it is contained in the fluid within the pustules. The disease can be directly inoculated from one person to another. Its poison can float in the air, can remain in rooms and clothing, can retain its vitality for long periods of time, and can be carried in clothing, etc., for thousands of miles. The susceptibility to the disease at all ages is very great, so that very few unvaccinated persons escape after exposure. One attack of the disease regularly protects against subsequent attacks.

*The period of incubation* usually lasts for from ten to fifteen days, although either longer or shorter periods are occasionally observed. During this period the patients may seem to be perfectly well; or they may suffer from loss of appetite, sleeplessness, and general malaise.

*The period of invasion* lasts for two or three days, rarely it extends to four, five, or six days. The invasion is sudden, with distinct chills or continued chilliness and a rapid rise of temperature up to from  $103^{\circ}$  to  $107^{\circ}$  F. The pulse becomes rapid and full, the breathing is rapid, there is marked prostration, the tongue is coated, the patients vomit, they may have general convulsions, they may become delirious. A severe aching pain in

the head and back are so constant and intense that they help to distinguish small-pox from the other infectious diseases. The mucous membrane of the tonsils and pharynx may be congested and dusky. The disease may prove fatal during the stage of invasion.

On the second and third days of the period of invasion are seen in some patients the so-called variolous rashes, which are not to be confounded with the regular eruption of the disease. These rashes appear in the form of points, streaks, or diffuse blushes, of red, purple, or brownish-red color. They are made paler by pressure, but do not show the white streak made by drawing the finger-nail over the inflamed skin, which is seen in scarlet fever. The surfaces involved are either not raised at all, or but slightly raised above the general level of the skin. The regions chiefly involved are the groin, the inner face of the thighs, and the hypogastric region, the axilla, the pectoral region, and the inner surface of the arm ; but the rash may involve any part of the skin. The rash may be mottled by hemorrhagic, petechial spots, or by large wheals ; it may appear on one part of the body, disappear, and recur in another place.

*The Period of Eruption.*—The eruption appears first on the face and scalp, and then extends so as to involve a large part of the skin and of the mucous membranes. At first it is in the form of rounded, red spots. By the second day of the eruption these spots have become papules ; by the sixth day they have changed into umbilicated vesicles ; by the eighth day the vesicles are changed into pustules. As the vesicles are changing into pustules the adjacent skin and mucous membranes become congested, swollen, and infiltrated with inflammatory products.

As the eruption first appears the temperature falls and the constitutional symptoms subside.

As the vesicles change into pustules and the skin becomes inflamed, the temperature rises, the pulse becomes more rapid, the patients suffer a great deal from the condition of the skin and mucous membranes, the prostration is very great, the patients are either stupid or delirious. In this condition the patients remain for from three to eight days.

Then, in the favorable cases, the temperature falls, the inflammation of the skin subsides, all the symptoms abate, the pustules dry up, scabs and crusts are formed, and finally the scabs fall off and leave cicatricial depressions in the skin. Five

or six weeks are required for the subsidence of the changes in the skin and the complete disappearance of all the constitutional symptoms.

*The confluent form of small-pox* is ushered in by a relatively short period of incubation, followed by a severe invasion. The chills are violent, the pains in the head and back are most severe, the temperature runs up to from  $105^{\circ}$  to  $110^{\circ}$  F.; the symptoms subside but little as the eruption appears.

The papules of the eruption are large and very close together. The vesicles and pustules are so close to each other that they coalesce, the skin is intensely inflamed and horribly swollen. The mucous membranes are much swollen, coated with mucus, pus, and false membranes, and sometimes gangrenous. The stench arising from the patient is intolerably fetid and pervading. The extensive suppuration of the skin is attended with the evidences of septic poisoning: the heart's action becomes rapid and feeble, delirium or stupor prevails, and the prostration is extreme. The patients are liable to complicating inflammations of the pericardium, pleura, bronchi, lungs, stomach, intestines, and kidneys.

*The malignant form of small-pox* is said to follow one of three types:

The first form has a short period of incubation, during which the patients suffer from a good deal of malaise and lumbar pain. On the fourth day there is a high temperature with a rapid pulse, speedily followed by a deep purplish-red staining of the face, neck, trunk, and extremities, the skin thus affected being slightly tumid and quite dry. At this time the eruption resembles that of black measles. Then papules are formed which rapidly change into large purpuric patches. The mucous surfaces become dry, cracked, and covered with crusts. There are hemorrhages from the larynx, bronchi, intestines, viscera, muscles, and serous membranes. The prostration from the first is extreme, the patients soon become unconscious, and death takes place within one or two days.

In the second form the period of incubation is also short. On the fourth day the skin is swollen and indurated in consequence of the presence of numerous firm papules very closely set together. These soon become larger and infiltrated with blood. Regular pustules are sometimes formed here and there, or parts of the skin may become gangrenous. Delirium, stupor, an in-

tense fever, and a rapid and feeble pulse continue, and the patients die within four or five days.

In the third form there is little rise of temperature, but the prostration and cerebral symptoms are equally marked ; the regular eruption is absent and replaced by an irregular rash or a few vesicles ; there is the same disposition to bleeding, and the same short duration and fatal termination of the disease.

*The hemorrhagic form of small-pox* is characterized by bleeding into the pustules and skin, and from the mouth, nose, bronchi, stomach, intestines, kidneys, and uterus. It has already been stated that this disposition to bleeding is especially marked in the malignant cases. It is also occasionally seen in the ordinary cases of small-pox.

*Small-pox modified by vaccination* (varioid) runs a milder and shorter course than does the unmodified disease.

The period of invasion may be shorter or longer, more or less severe, than that of true small-pox. It may be accompanied by the variolous rashes. With the appearance of the eruption the subsidence of the constitutional symptoms is nearly complete. The eruption is not very extensive, the pocks are small, some of the vesicles dry up without suppurating, the pustules are not large or deep, there is little diffuse inflammation of the skin or mucous membranes ; desiccation takes place on from the fifth to the seventh day of the eruption.

The patients regularly recover, and some are not sick enough at any time to be in bed.

Such a modified small-pox, however, is capable of giving true small-pox to persons unprotected by vaccination.

*The convalescence* may be interfered with by boils, erysipelas, gangrene of the skin, laryngitis, pleurisy, pneumonia, myelitis, or septicæmia. The eye-sight and the hearing are often completely destroyed by the pustules, the skin is permanently deformed by the cicatrices left by the pustules.

*Mortality.*—In the last century, in many countries, from seven to twelve per cent. of all the deaths were due to small-pox. Since the introduction of vaccination in the same countries small-pox is a comparatively rare disease.

Malignant and hemorrhagic small-pox are almost certainly fatal ; confluent small-pox is very dangerous to life ; from discrete small-pox the percentage of recoveries is considerable ; varioid is very seldom fatal.

*Prophylaxis.*—The most important duty of the physician as regards small-pox is his insistence on universal vaccination and revaccination. Where these are thoroughly carried out small-pox is an infrequent disease; where they are neglected serious epidemics attack large numbers of people.

*Treatment.*—The patients are to be kept in bed with the ventilation, feeding, and nursing appropriate to all the severe infectious diseases.

The skin is to be covered with either cold or warm compresses of a weak solution of carbolic or boric acid, and frequently bathed; or frequent immersions in warm baths for several hours every day may be practised.

*Vaccination.*—The great mortality from small-pox naturally led to attempts to protect the community against its ravages. It was known that one attack of the disease protected against subsequent attacks, and so the trial was made of inoculation of matter from the pustules of small-pox. It was found that the protection afforded by inoculation was efficient, but that its practice was attended with inconvenience and danger.

Then the discovery was made that there was a disease of cattle—cow-pox—which resembles small-pox, and that the virus from cow-pox, when inoculated in human beings, gave them an immunity from small-pox. From the time of this discovery vaccination has become an established practice, and small-pox has lost its terrors.

When vaccination is practised in an unvaccinated person, on the third day a papule is formed; from the fifth to the ninth day a vesicle is formed on the papule; on the eighth day the vesicle and papule become the seat of suppurative inflammation; then the inflammation subsides, and from the twentieth to the twenty-fifth day the scab falls off and leaves a cicatrix in the skin. The evolution of the pustule is accompanied with slight constitutional disturbance.

When vaccination is practised in a person who has already been vaccinated, there may be nothing effected, or the formation of a localized inflammation of the skin, or an irregular vaccine pustule. The ordinary rule is to vaccinate all infants, to revaccinate once in about seven years, or oftener in case of exposure.

The protection afforded by vaccination varies in different individuals as to its length of time and its completeness. It may

afford absolute immunity against small-pox, or it may only insure a mild form of the disease.

The only dangers attending vaccination are erysipelas, septicæmia, and syphilis. All of these can be guarded against. It is customary to use the virus taken from the pustules of infants or from the pustules of calves.

### MEASLES.

Measles is a very contagious disease. Children are much more susceptible to it than are adults. It is contagious both in the period of invasion and in that of eruption. The poison of the disease is given off from the surface of the body, it floats in the air, it can be inoculated with the blood, it lodges in clothing, bedding, etc.

Among the tenement-house population, in asylums, and in armies the disease is often of severe type and fatal of itself, or by its complications. When introduced for the first time in savage tribes it is exceedingly fatal. But as we see the disease under ordinary conditions, it is of mild type and very seldom fatal.

*Lesions.*—The characteristic lesion is the eruption which appears on the skin and mucous membranes.

The accessory lesions are : the catarrhal inflammations of the mucous membranes of the eyes, nose, throat, larynx, bronchi, stomach, and intestines ; the croupous laryngitis ; broncho-pneumonia ; simple, suppurative, or tubercular inflammation of the bronchial or cervical lymphatic glands ; suppurative otitis ; acute degeneration of the kidneys, or acute exudative nephritis.

The period of incubation after inoculation with the blood is seven days ; after ordinary exposure, from ten to fourteen days. During the period of incubation there may be a little fever and disturbances of digestion.

The period of invasion lasts from twelve hours to six days, usually four days.

It is marked by chills, sometimes general convulsions, a rapid rise of temperature, headache, vomiting, and diarrhœa. At the same time are added the symptoms of the catarrhal inflammation of one or more of the mucous membranes. The cases vary as to the number and severity of the symptoms during the period of invasion.

The eruption is of maculo-papular form tending to the cre-

scentic shape. The patches are of light or dark red color, of crescentic or irregular shape, scattered or confluent, flat or papular, sometimes covered with vesicles, sometimes hemorrhagic. There is more or less inflammation of the skin and mucous membranes between the blotches of the eruption.

The eruption appears on the face, neck, trunk, and extremities, and on the mucous membranes. It reaches its full development in from two to four days.

After from ten to fourteen days there is a fine desquamation.

The fever increases as the eruption is developed, and is usually highest as the eruption becomes complete.

At the same time there are often cerebral symptoms : restlessness, sleeplessness, stupor, delirium, convulsions.

During the period of eruption the patients suffer from the symptoms which belong to the inflammation of one or more of the mucous membranes.

The conjunctivitis is attended with pain and intolerance of light.

The pharyngitis causes pain in swallowing and cough.

The laryngitis gives pain, laryngeal cough, laryngeal voice, laryngeal dyspnœa.

With the bronchitis we find the ordinary symptoms and physical signs.

The gastritis produces pain and vomiting ; the enteritis, pain and diarrhœa.

The eruption and the fever regularly subside in from two to seven days, desquamation follows, and convalescence is established from the tenth to the fourteenth day after the beginning of the eruption.

*The complications* of most consequence are : broncho-pneumonia, gangrene of the mouth and vulva, suppurative otitis, adenitis, croupous laryngitis, and nephritis.

*The course of the disease* is regular, or very mild, or severe, or malignant.

The malignant cases are sometimes called "black measles." The eruption is imperfectly developed, but of very dark color ; the prostration is extreme, with rapid and feeble heart-action, a cold skin, and a brown and dry tongue ; there are marked cerebral symptoms and bleeding from the mucous membranes.

*Convalescence* may be interrupted by persistence of the conjunctivitis, pharyngitis, otitis, bronchitis, broncho-pneumonia,

gastritis, or tubercular adenitis. Or the patients may be left for a long time feeble and anæmic.

*The prognosis* in ordinary cases is very good. It is unfavorable in children under two years of age, in adults, in countries where the disease prevails for the first time, in armies, in asylums, and in tenement-houses.

*Treatment* is directed to the fever, the cerebral symptoms, the skin, the conjunctivitis, pharyngitis, laryngitis, bronchitis, broncho-pneumonia, gastritis, enteritis, and adenitis.

#### VARICELLA.

Chicken-pox.

This disease is almost confined to children, but yet is occasionally seen in adults.

It is fairly contagious, the poison is given off from the surface of the sick body.

The period of incubation is from thirteen to seventeen days.

The period of invasion lasts for one or two days. The patients have a little fever, disturbances of digestion, and slight prostration.

The period of eruption lasts from two to five days. First there are little rounded red spots in the skin, then papules, and then vesicles. Many of the vesicles shrink and dry up, a few go on to form pustules. The vesicles, when they have run their course, leave the skin smooth; the pustules leave little cicatricial depressions.

The eruption appears on the skin, scalp, mouth, and pharynx.

The disease is a very mild one, and in many cases there can hardly be said to be any except the symptoms eruption.

#### SCARLATINA.

*Lesions.*—The characteristic lesion is the eruption.

The accessory lesions are frequent and important.

The pharynx and tonsils are the seat of catarrhal, croupous, or gangrenous inflammation.

The larynx may be invaded by the croupous inflammation of the pharynx.

The Eustachian tube is often the seat of catarrhal inflammation, the middle ear of suppurative inflammation.



The cervical lymphatic glands may be inflamed and swollen, or they may suppurate. The kidneys are regularly the seat of acute degeneration, of acute exudative nephritis, or of acute diffuse nephritis.

*Etiology.*—The specific micro-organism of scarlet fever has not yet been determined. The poison seems to exist on the surface of the body of the sick person, in the desquamated epidermis, and in the blood. It is not carried to any distance by the air, but it adheres tenaciously to articles of clothing, furniture, toys, etc., and retains its vitality for a long time. The disease is, therefore, easily carried about from place to place. It can be inoculated with the blood, but we do not know certainly the ordinary way in which the disease enters the body. Although the poison is an active one and exposure is regularly followed by infection, yet a considerable number of persons enjoy throughout their lives entire immunity from the disease, no matter how thoroughly they are exposed. The greatest liability to the disease is in children between the ages of two and seven years. Adults are much less susceptible to the disease.

The puerperal condition and surgical operations sometimes seem to render persons more liable to the disease than they otherwise would be. One attack regularly, but not always, protects against subsequent attacks. The disease varies greatly in its severity at different times and in different places.

*The period of incubation* is regularly seven days. The extreme limits of this period are twenty-four hours, and three weeks. There are usually no symptoms during the period of invasion, but occasionally there is a little sore throat and the patients do not feel quite well.

*The period of invasion* lasts for from twelve to forty-eight hours, but is occasionally prolonged for several days. It is marked with a sudden rise of temperature, accompanied at first with chills. The height reached by the temperature within the first twenty-four hours is in proportion to the severity of the disease. With the fever come pains in the head and back, prostration, restlessness, delirium, and stupor.

In addition to the fever the invasion is attended with general convulsions, sore throat, or vomiting.

The convulsions are especially common in children, there may be one convulsion or several within the first twenty-four hours.

The vomiting belongs to the first hours of the invasion, either a single sudden attack, or repeated vomiting and retching.

The sore throat is a general catarrhal inflammation of the pharynx and tonsils. It is attended with a good deal of congestion, so that the throat looks more red and angry than is the case with a simple pharyngitis. With the sore throat there is also a swelling of the papillæ of the tongue, the strawberry tongue.

The fever increases, the sore throat, the cerebral symptoms, and the prostration continue while the eruption is developing.

*The eruption* is in the form of minute red points. These are numerous and close together, so as to form an extensive diffuse redness; or they are aggregated in irregular patches; or they are separated and scattered. The eruption first appears on the neck and shoulders, and then extends to the trunk, arms, and legs. It reaches its full development in from one to four days, and then the skin is nearly uniformly red, swollen, and tense. The regular and fully developed eruption is very characteristic and not likely to be mistaken for that of any other disease. But irregular forms are of common occurrence, and are often very puzzling. The eruption may be confined to the trunk, the limbs, or the face; it may never reach its full development, but remain in the form of scattered patches or points; its entire duration may be confined to a few hours; there are cases in which no rash is seen at any time.

The whole duration of a regular eruption is from three to ten days. As the eruption subsides the dead epidermis begins to come away in flakes and large patches, and this desquamation continues until the end of the third week, or even longer.

*Course of the Disease.*—1. The regular cases.

The invasion of the disease is sudden, with chills, fever, headache, restlessness, delirium, and, in addition, vomiting, convulsions, or sore throat. After from twelve to thirty-six hours, while the temperature continues to rise and the patients begin to look more sick, the eruption appears. It is seen first on the neck and chest, then rapidly extends over most of the body, and the skin becomes uniformly red and swollen. By the fourth day the inflammation of the skin reaches its maximum, and after that day subsides. In from five to ten days the eruption has entirely disappeared. About the seventh day the pharyngitis subsides, the temperature falls, all the constitutional symptoms disappear. By the end of

the second week the patients seem to be well. By the end of the third week desquamation is complete and the disease has run its course.

2. The disease runs its regular course, but the fever, instead of subsiding on the seventh day, continues for one or two weeks.

3. The eruption is irregular as to its appearance, situation, or duration, while the other symptoms are regular.

4. There are very mild cases with but trivial constitutional symptoms and a scanty and short eruption, but with the characteristic desquamation and the liability to complications.

5. There are cases in which the only symptoms are fever and sore throat.

6. There are severe and prolonged cases. The eruption is either scanty or abundant, but the constitutional symptoms are marked, especially the fever and cerebral symptoms. The patients are seriously ill for two or three weeks, and may die from the scarlatina alone without any complication.

7. There are malignant cases. The patients look from the first as if they were overwhelmed with the poison of the disease. The temperature is excessively high or below the normal. The cerebral symptoms are marked and the heart's action rapid and feeble. Vomiting, diarrhœa, dyspnœa, and bleeding from the mucous membrane are often present. The eruption is irregular and dark-colored. The patients die in from eight hours to two days.

Dr. H. Noble Joynt (*Lancet*, 1891) gives the following description from the personal observation of one hundred malignant cases :

Although the general course is similar in all instances, yet clinically three well-marked varieties may be noted, as regards the character of the rash, the extent of the throat inflammation, the prominence of the nervous symptoms, the duration of the fever, and the prognosis.

In the first or asthenic variety the following characteristics appear: The rash is badly marked, being either wholly suppressed or scanty, distributed in irregular dark blotches on the limbs and gluteal region ; or, again, it may consist of a general eruption of scattered dark maculæ resembling typhus fever. Purpuric blotches and spots are frequent, as well as petechiæ on the arms, chest, sides, back, and abdomen, but best seen over the legs and backs of the feet. The lower extremities are cold and pre-

sent a livid appearance. The face is pale, pinched, and cyanosed, the lips and cheeks bluish, and the circumoral pallor pronounced. The eyes become sunken and surrounded by dark rings; the conjunctiva is sometimes injected, but usually anæmic; the cornea dull and covered with a mucous film; the eyelids fail to close. At first the tongue is thickly furred, but becomes dry and coated with a dirty-brown secretion; sordes gather on the lips and teeth. The fauces are somewhat swollen, foul, dryish, of a dark purplish-red color, and the tonsils slightly enlarged. Unless kept frequently syringed all the structures of the mouth become coated with a foul viscid mucus, which clings to the palate and tongue, and effectually hinders inspection of the fauces. If the throat inflammation becomes more intense, the tonsils enlarge, superficially ulcerate, and a fetid, purulent discharge oozes from the nostrils. But in this group ulceration is always slight and secondary, faucial swelling and dark congestion being the conspicuous features. Vomiting of a green, bilious nature is very frequent, the irritability of the stomach is extreme, and nothing swallowed is retained. To add to the difficulty of feeding the patient, diarrhœa accompanies the vomiting and renders the rectal alimentation wellnigh impossible. The temperature is always high, ranging from 102° F. to 106° F. The pulse is very rapid, reaching 200 beats per minute; it is soft, irregular, compressible, and so feeble as to be imperceptible at the wrist. The heart is excited, quickly becomes weak, the sounds barely distinguishable, and in young children uncountable. Respiration is quickened, shallow, and sighing, and toward the end may exhibit the Cheyne-Stokes rhythm. The nervous symptoms are among the most pronounced. Delirium, worse at night, is constant, often continuing throughout the whole illness. At first the patient may be violent, getting out of bed, etc.; more usually, however, the delirium is of a low muttering type, gradually passing into coma. The child is very restless, rolling from side to side, tossing his arms about, boring his head into the pillow, moaning, and grinding his teeth. Muscular tremors of the tongue, face, and hands, and twitching of single groups of muscles may be noticed; but, above all, what strikes the observer is the utter prostration. Toward the end the patient sinks into a comatose condition. If he lives four or five days, a typhoid state, with dry tongue, cold extremities, dull eyes, foul mouth, sordes, pinched, pallid face, involuntary evacuations, hiccough,

etc., develop. In young adults the mind may continue clear almost to the last. Syncope is a common cause of death ; convulsions in a few instances bring on the fatal ending. Death occurs as a rule within four days ; in young, weakly, and rickety children in a shorter period. One boy, aged two, whose sister also died of malignant symptoms, succumbed in thirty hours ; a girl, aged three, in thirty-six hours ; another boy, aged fourteen, in about fifty-six hours ; and sixteen others died in seventy-two hours and under. As a rule, the older and stronger the subject, the better the fight for life is. Thus a man, aged twenty-eight, lingered for six days ; another, aged eighteen, lived for five ; and a third boy, aged seventeen, had sufficient vitality to pull through, but when apparently convalescing well, fell a victim to an unexpected attack of double basal pneumonia during the third week.

In the second or anginal variety the character and course of the fever are much the same as that already described, but the throat symptoms are more conspicuous. The tonsils are more swollen, ulceration is always present, may spread to the pharynx and nasal passages, and set up a fetid, purulent rhinorrhœa. A diphtheritic membrane often coats the tonsils and meatus of the nose. The mouth is very foul, and the submaxillary glands enlarge to a moderate degree. The rash is scanty, dark, patchy, petechial, often of a livid hue, especially on the extremities ; rarely is the eruption general, and then it is measly and dark. Delirium and vomiting are not so marked, but the temperature is as high, and the pulse as rapid and feeble, as in the asthenic variety. Slight laryngitis was noted in two instances on the second and third days, respectively. This group presents a likeness to the acuter course of anginal scarlatina, and indeed is the connecting link between the two. But it differs from the true anginal fever in that there are no secondary inflammatory or plastic lesions, such as adenitis, cellulitis, otitis media, or other extensive ulcerative changes, in the pharynx and nasal passages ; moreover, death is due to the toxic effects of the scarlatinal poison on the nerve-centres, and not to exhaustion from suppuration, septic infection, or inflammatory obstruction of the vital organs. It may be laid down as a general rule that in scarlatinal angina death is caused by a complication. In this type children under five, with short necks and strumous or rickety habit, are the chief sufferers, and death is frequently the result of syncope.

The third or congestive variety presents some remarkable differences from the foregoing. The eruption is very characteristic. The whole body is covered with a dark, often livid, intense diffuse or punctiform rash, aptly likened to the color of a boiled lobster. Petechiæ, small hemorrhages, and darker maculæ are common. The face is flushed, the rash well developed over the scalp, on the forehead, and sides of the face, and the circumoral pallor is replaced by a crimson hue. The eyes are considerably congested, and hemorrhages may take place into the orbital conjunctivæ. A copious miliary eruption on the trunk is sometimes seen. About the fourth day the skin becomes dry, often leathery or parchment-like in texture, and of a dark red-brown color. At the same time desquamation appears on the forehead and cheeks, round the nose and mouth, on the chin, neck, shoulders, and inside of thighs. The skin peels off in sheets, and with care casts of the fingers and toes may be obtained. In cases that recover the skin may be completely cast off twice or thrice in the first fortnight. The secondary skin, and even the third, has the intense scarlet hue of the primary rash. The throat is dry, but little swollen; the tonsils are seldom enlarged, but the mucous membrane has a peculiar dark red-brown hue, well likened by Dr. W. H. Line\* "to the appearance and glazed lustre of well-polished Spanish mahogany;" ulceration is uncommon, and superficial when present. The tongue is pointed and dark, raw-looking, stripped by the third day, and the papillæ stand out prominently. Active delirium is present in nearly every case, and it is often difficult to keep the patient in bed. Vomiting is infrequent, but before death the stomach may reject everything. The temperature ranges from 102° to 106° F. and higher; the pulse is rapid, soft, and compressible. As the fever advances nervous tremors develop, prostration becomes very marked, the eyes become sunken, the face pinched and cyanosed, the pulse thready, hypostatic congestion of the lungs sets in, the motions are passed involuntarily, and death ensues from the fourth to the eighth day. Fortunately, however, this variety is not so fatal as the other two; three-fifths of our cases recovered. This may, perhaps, be accounted for by the fact that the patients were older and stronger than in the former classes. It is more common in patients over seven years of age. Thus, in 25 cases, 7 occurred in patients under five, of whom 5 died; 8

\* Birmingham Medical Review, March, 1885.

between six and ten ; 7 between eleven and fifteen ; and 3 over sixteen. As regards sex, 14 were males and 11 females.

8. A person suffering from scarlet fever may, at any time in the course of the disease, have an attack of true diphtheria.

9. Persons suffering from scarlet fever, instead of having the ordinary catarrhal inflammation of the pharynx and tonsils, may have a croupous inflammation with the growth of streptococci. These patients are said to have scarlatinal diphtheria, but the inflammation is accompanied by the growth of streptococci, not by the growth of the Klebs-Löffler bacillus. Such a croupous inflammation may be of mild or severe type.

(a) On the first, second, or third day of the scarlet fever, one or both tonsils become swollen and coated with small patches of false membrane which gradually increase in size, and the lymphatic glands of the neck become somewhat swollen. The inflammation continues for about four days, then subsides, and by the end of a week has disappeared. While the inflammation of the throat is running its course, the symptoms of the scarlet fever continue and are not changed by the sore throat, nor is the patient in any danger from it.

(b) The mucous membrane of the tonsils and pharynx is at first intensely congested and swollen. About the third day of the scarlet fever the temperature runs up and the mucous membrane of the throat and nose is much more swollen, covered with tenacious mucus, and a foul-smelling fluid exudes from the nose. The lymphatic glands in the neck are swollen, and soon the connective tissue of the neck is swollen, hard, and infiltrated, and this swelling increases, pushes the head backward, and extends around the neck. The patients now look as if they were suffering from septic poisoning, with high temperatures and a feeble heart. Next comes the necrosis of portions of the inflamed mucous membrane, of the lymphatic glands, and of the infiltrated tissues of the neck, and soon after this the death of the patient. The whole picture is that of a gangrenous inflammation with septic poisoning.

(c) The scarlet fever begins without any alarming symptoms, the tonsils and pharynx are moderately inflamed, the patients seem to be doing well until the latter part of the first week of the disease. Then, instead of beginning to improve, the patients seem to stand still or to look a little worse. The throat is still only moderately inflamed, but the lymphatic glands in the neck

are swollen. After this the mucous membrane of the throat becomes a little more swollen, with more or less false membrane, a yellow or bloody fluid exudes from the nose, there are unhealthy fissures at the edge of the nose and the corners of the mouth. It is not, however, until the second week of the scarlet fever that the condition of these patients becomes threatening. The lymphatic glands in the neck are now much enlarged, with infiltration of the adjacent connective tissue and skin. There is a discharge of foul-smelling saliva from the mouth, ulcers are formed in the pharynx and tonsils with gangrenous edges, and brown, gangrenous patches can be seen in the mucous membrane. The lymphatic glands and the skin of the neck also become gangrenous, the large veins and arteries in the neck may be eroded, or septic thrombi are formed in the veins. The patients may have diarrhœa, or purulent inflammation of the joints, or purulent inflammation of the serous membranes. The temperature continues high, the patients steadily lose flesh and strength, and die after from three to five weeks.

10. Suppurative inflammation of the middle ear may be developed at any time in the course of scarlet fever. It is attended with a rise of temperature, pain, restlessness, sometimes stupor and delirium. These symptoms may be so marked as to simulate an acute meningitis. They continue until the membrana tympani is ruptured and the pus escapes.

11. Inflammation of the lymphatic glands of the neck belongs to the middle or end of the second week. The glands are simply swollen or they suppurate. Such an adenitis is accompanied with fever and sometimes lasts for a long time.

12. Acute pericarditis or endocarditis may be developed at any time in the disease.

13. Inflammation of the joints or of the muscles may occur as the eruption is subsiding. The joints most frequently affected are those of the hands and feet; the muscles, those of the neck. With the synovitis or myositis there is more or less fever.

14. The poison of scarlet fever is capable of producing three morbid changes in the kidneys:

(a) Acute degeneration belongs to the first and second weeks of the disease. It is of mild type. The quantity of the urine is but slightly diminished, its specific gravity is unchanged, it contains a little albumin and a few casts. It is not accompanied with any constitutional symptoms, and the kidneys return to



their normal condition after the patients have recovered from their scarlet fever.

(b) Acute exudative nephritis belongs to the second and third weeks of scarlet fever. The urine is scanty or suppressed, its specific gravity is unchanged, it contains large quantities of albumin, numerous casts, and blood. The temperature rises sometimes as high as  $105^{\circ}$ ; there are prostration, headache, nausea, vomiting, anæmia, and dropsy. In some of the patients there is added contraction of the arteries, with disturbance of the heart's action and general convulsions, delirium, or stupor. Most of these cases of nephritis last about four weeks, and terminate in recovery. A smaller number prove fatal.

(c) Acute diffuse nephritis belongs to the third week and the period of convalescence. It follows either an acute or subacute type. The acute cases in their early stages resemble exudative nephritis. In the subacute cases the symptoms are developed gradually. The urine is somewhat diminished in quantity, contains much albumin and casts, but no blood. There is no fever. The anæmia and dropsy are developed gradually. In all cases of diffuse nephritis the kidney disease is apt to persist and to go on, quickly, slowly, or with intermissions, up to the patient's death.

15. In the third week of scarlet fever there may be developed a general subcutaneous œdema without disease of the kidneys. This seems to depend in some way on the inflammation of the skin.

16. Meningitis, pleurisy, broncho-pneumonia, and peritonitis are comparatively rare complications.

*Convalescence.*—Some of the patients are left, after the scarlet fever, for a long time feeble and anæmic, or mentally feeble.

Chronic nephritis, protracted adenitis, or protracted endocarditis may seriously interfere with convalescence.

*Treatment.*—The patients must be kept in one room, or one set of rooms, for three weeks from the beginning of the eruption. They must be kept in bed while the fever and constitutional symptoms continue. They are not to be allowed animal food until the completion of the three weeks. The rooms are to be kept well ventilated, the patient's skin must be washed every day with warm water and soap.

Excessively high temperatures may be reduced by antipyrin, antifebrin, or phenacetine.

The sleeplessness, restlessness, and cerebral symptoms may be alleviated by opium, the bromides, or sulphonal.

Special treatment may be needed for the pharyngitis, otitis, adenitis, or nephritis.

#### GERMAN MEASLES.

*Synonyms.*—Rötheln ; Roseola ; Rubella ; Rubeola.

*History.*—During the eighteenth and nineteenth centuries, in different countries, there have been numerous epidemics of eruptive diseases which resembled either measles or scarlet fever, and yet seemed in some ways distinct from these diseases. So two opinions have been held on the subject. Some physicians have believed that there is an exanthematous disease entirely distinct from measles and scarlet fever ; others that the apparent examples of such a disease are only irregular forms of the two well-known exanthemata. At the present time the prevailing opinion seems to be that there is a distinct exanthematous disease apart from measles and scarlet fever which is not a modification or a variety of either of these diseases.

*Etiology.*—The disease seems to be communicated from person to person, but the poison is not very active nor very long lived, so that of all the persons exposed a considerable number escape. It occurs for the most part in epidemics, and may disappear altogether between the epidemics. Children are more liable to the disease than are adults. The epidemics of German measles have often occurred at the same time with an increased prevalence of true measles, or of scarlet fever, and it has been found that German measles does not protect against either measles or scarlet fever, but does protect against itself.

*The period of incubation* seems to vary in different epidemics ; the most frequent length of time is from fourteen to twenty-one days, but an incubation of only six days is not uncommon. There are no symptoms during this period.

*A period of invasion* is very often entirely absent. When it does occur it does not usually last more than twenty-four hours, but it may be prolonged for two, three, four, or even five days. The symptoms which belong to this period are : a febrile movement, headache, rarely general convulsions, nausea, vomiting, conjunctivitis, pharyngitis, laryngitis, and swelling of the glands of the neck. As compared with measles or scarlet fever, the symptoms of the invasion of this disease are insignificant.

*Period of Eruption.*—Very often the patient is ignorant of his attack until he discovers the eruption, and this discovery is often made early in the morning. The rash appears first on the forehead and temples, rapidly extends over the face, neck, trunk, and lastly over the extremities. It is in the form of minute pinkish points, which are aggregated into separate blotches like those of measles, or into a diffuse rash like that of scarlet fever.

In some cases the maculæ are changed to papulæ, and on the surfaces of these are small vesicles or pustules. The eruption is regularly the prominent symptom, altogether out of proportion to the constitutional symptoms. It appears on the face, body, and extremities, and lasts for from one to seven days. It often fades in one place before it appears in another. It is not usually followed by desquamation.

During the period of eruption there may be a little fever, a coated tongue, conjunctivitis, pharyngitis, swelling of the lymphatic glands in the back of the neck, synovitis of the smaller joints, inflammation of the muscles of the neck, bronchitis, or broncho-pneumonia.

The disease is not a severe one, and requires no treatment except for the alleviation of symptoms.

## THE MALARIAL DISEASES.

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This name is given to a group of disorders which are produced by a special poison originating in the earth and taken into the bodies of human beings. These disorders are called Intermittent Fever, Remittent Fever, and Malarial Cachexia.

The malarial diseases exist in their most severe forms in Africa, Asia, India, China, the West India Islands, South America, and Central America. In these countries remittent fever and malarial cachexia prevail constantly, are of severe type, and are often fatal.

These diseases exist in a well-marked form in the southern and western portions of the United States, in parts of Russia, Greece, Hungary, Italy, Germany, France, the Netherlands, Spain, England, and Scotland. In these countries intermittent fever, remittent fever, and malarial cachexia prevail nearly constantly, occur in epidemics, and exhibit varying degrees of severity. The disease will from time to time invade fresh districts, or will assume the mild form, or may disappear altogether from regions where it has already prevailed.

The mild form of malarial disease is seen in the middle and eastern portions of the United States and in parts of Europe. Intermittent fever and malarial cachexia in their milder forms come and go in different localities, sometimes very prevalent, sometimes rare, sometimes disappearing altogether.

In the countries where the most severe form of malaria exists, the rainy season is the worst.

In those countries where its well-marked form prevails, it is most common in spring and autumn.

In those places where the mild form of the disease belongs, it is most common in spring and autumn.

The ordinary rules are: That the frequency of the disease diminishes with the elevation above the sea-level; that marshy soils are the worst and sandy soils the best; that the draining of marshes and ponds, the cultivation of virgin soil, and the aban-

donment for a long time of cultivated soil, are followed by an increased prevalence of the disease.

Where the mild forms of the disease exist it is difficult to lay down any rules as to the conditions which favor the appearance and disappearance of the disease.

There seems to be a well-marked difference in different individuals as to their susceptibility to the disease.

*Lesions.*—The poison of the malarial diseases is a form of protozoa, called the “*plasmodium malariae*.” The organism seems to originate, live, and grow in the earth and water, and to be capable of floating in the air. It is apparently taken into the human body through the lungs. After they have been taken into the human body the micro-organisms find their way into the red blood-cells, they increase in size, they multiply, they destroy the red blood-cells with liberation of their pigment.

In its earliest form the plasmodium is in the form of very small, pale, crescentic, or irregular bodies contained within the red blood-cells. These bodies increase in size, the red blood-cells are destroyed, their pigment is set free, and is found in blackish granules in the plasmodium, in the white blood-cells, and in free masses and granules.

The pigment is especially abundant in the blood of the liver and spleen. Some of the organisms assume a peculiar shape, as of a body with long threads projecting from it. After a time there is a segmentation of the organisms and the formation of a new crop, which again invade the red blood-cells.

The spleen is very often hypertrophied. The red blood-cells are diminished in number. The pigment due to the destruction of the red blood-cells may be found within the blood-vessels in all parts of the body, but in the liver and spleen the quantity is so great that these organs are fairly black.

#### INTERMITTENT FEVER.

The period of incubation varies from a few days to several months.

When the disease has once been acquired, the paroxysms may return at intervals, without fresh exposure, for many years. Change of climate and accidental circumstances may be the exciting causes of paroxysms.

The symptoms occur in paroxysms, which are repeated at

regular intervals, every day, every other day, or at longer intervals. The paroxysms are repeated at the same hour on the successive days, or the time may be anticipated or postponed.

Each complete paroxysm has three stages: the cold, the hot, and the sweating.

The cold stage is attended with chills, a cold skin, a rapid pulse, and irritability of the stomach. It lasts for from half an hour to two hours.

The hot stage is attended with a rapid rise of temperature, the skin is hot and dry, the pulse full and rapid; vomiting, headache, and pain in the back are present. It lasts for from two to twelve hours.

In the stage of sweating, the temperature falls, all the symptoms subside, and there is profuse perspiration.

Irregular paroxysms may occur, in which either the cold or the sweating stage are absent.

When the paroxysms are repeated for a long time, the spleen is hypertrophied, the number of red blood-cells is diminished, and the general health is impaired.

Instead of a regular paroxysm, there may be a chill, a rise of temperature, and the passage of pigmented urine (intermittent hæmatinuria).

*Pernicious Intermittent.*—1. After the chill, instead of the fever there is feeble heart-action and collapse.

2. In the third stage there is profuse sweating and collapse.

3. During the chill there is dangerous heart-failure.

4. In the second stage there is coma or violent delirium.

5. In the second stage there is congestion of the lungs, with cough, dyspnœa, and profuse mucous expectoration.

6. In the second stage there is congestion of the stomach, with pain, vomiting, and hæmatemesis; or congestion of the intestines with pain, purging, and bloody stools.

*Treatment.*—The inhabitants of malarial districts learn to select the situation of their houses, to look after their drainage, and to avoid the night air.

Persons who have to pass a short time in a malarial district do well to take from six to ten grains of quinine after breakfast every morning.

To abort a paroxysm we may give a hypodermic of one-fifth of a grain of muriate of pilocarpin, and one-eighth of a grain of

sulphate of morphine, together, or a full dose of opium, or drachm doses of chloroform, or antifebrin, or antipyrin.

To cure the disease we may use quinine. The ordinary dose is from twelve to twenty grains between the paroxysms. The efficiency of the quinine is much increased by giving it in solution. In bad cases the dose must be larger.

In some cases we do better with Warburg's tincture in capsules, four to eight capsules a day.

In some cases there is an advantage in adding arsenic to the quinine.

In all cases it is the rule to continue the treatment for some weeks after the paroxysms have stopped.

In some cases the paroxysms cannot be stopped except by change of climate.

#### REMITTENT FEVER.

The invasion of the symptoms may be sudden, or preceded by one or more paroxysms of intermittent fever, or by a prodromic period of several days' duration.

The invasion of the disease is marked by one or more chills, more or less severe. The temperature begins to rise during the chills. The patients suffer from headache, pains in the back and limbs, a rapid pulse, a coated tongue, nausea, and vomiting.

There is a remission of the fever in the early morning, and sometimes a second remission in the evening. The bowels are constipated. The urine is acid and high-colored.

The fever lasts for from five to twenty-one days; it terminates in recovery, or in death, or is followed by intermittent fever.

In the mild cases the patients are not at any time very sick, and regularly recover.

In the severe cases the temperature is high and nearly continuous, the tongue becomes dry, the pulse is rapid and feeble, the patients pass into the typhoid state, with delirium and diarrhœa.

In the malignant cases the prostration, the cerebral symptoms, the jaundice, and the hemorrhages from the mucous membranes are marked symptoms.

*Treatment.*—At the commencement of the disease it is customary to give first a mercurial purge and then quinine, or Warburg's tincture. If these are of service their use is continued;

if they exert no effect on the disease they are discontinued, and we fall back on the nursing and the treatment of symptoms, until the time for the subsidence of the disease, when the quinine may be tried again.

#### MALARIAL CACHEXIA.

Either with or without previous attacks of intermittent or remittent fever, persons may become the subjects of malarial cachexia.

The essential feature of malarial cachexia seems to be the destruction of red blood-cells and the consequent anæmia with its attendant symptoms. There is also regularly hypertrophy of the spleen.

The patients become pale, feeble, languid, and emaciated. They suffer from headache, neuralgic pains, and disturbances of digestion.

The condition of these patients is often very miserable, and it is possible for them to die with extreme anæmia or leukæmia.

*Treatment.*—The most important point in the treatment of these patients is their removal from the malarial district to one where no malaria exists. In addition we employ arsenic, iron, and measures likely to improve the digestion and the nutrition.

#### THE MALARIAL DISEASES OF NEW YORK.

1. *The Intermittent Type.*—(a) The patients have regular and complete paroxysms of intermittent fever.

(b) They have paroxysms of fever alone, the temperature usually not over 100° F., at regular or irregular intervals, in the evening or at night.

(c) They have paroxysms without fever, but with malaise, headache, sleeplessness, irritability of temper, palpitation of the heart, loss of appetite, nausea, disturbances of the functions of the liver, and abnormal sensations. The paroxysms come on in the evening or at night, at regular or irregular intervals.

2. *The Remittent Type.* (a) The patients have a morning temperature of about 100° F., and an afternoon temperature of from 104° to 106° F. They do not look very sick. I have seen a number of such cases in children. In women after childbirth such a fever is not infrequent. It may give rise to much alarm and to fears of pelvic inflammation. It is a curious feature of this fever



that, while in some cases it yields promptly to quinine, in other cases it continues in spite of all treatment until the patients are taken out of New York.

(b) The patients have fever,  $103^{\circ}$  to  $104^{\circ}$  F., with headache and moderate prostration, for two weeks, and then get well without further trouble.

(c) The patients have a little fever,  $99^{\circ}$  to  $102^{\circ}$  F., higher every evening. This fever lasts for weeks and months. The patients are not usually in bed, but feel very miserable. They have headache, disturbances of digestion, abnormal sensations, lose flesh and strength.

3. *The Malarial Cachexia*.—This follows the ordinary type of the milder forms of malarial cachexia.

*Treatment*.—There is a very great difference in different patients as to the effect of remedies. Quinine, arsenic, or Warburg's tincture act quickly and efficiently for some, while for others they are absolutely useless. Attention to the digestion, the general health, and change of climate are always necessary.

## WHOOPING-COUGH.

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(PERTUSSIS.)

An infectious disease characterized by inflammation of the respiratory tract and a peculiar paroxysmal cough.

*Lesions.*—There is catarrhal inflammation of the nose, larynx, trachea, and bronchi, sometimes broncho-pneumonia.

The poison of the disease has not been demonstrated. It is apparently given off with the breath, floats in the air, and is taken into the bronchi.

The disease is contagious from person to person. One attack protects against subsequent exposure. It often accompanies epidemics of measles.

The period of incubation lasts for two weeks.

The invasion begins with the symptoms of the inflammation of the nose, larynx, trachea, and bronchi, in varying degrees of severity. The bronchitis is the prominent symptom. These symptoms last for one or more weeks before the appearance of the paroxysmal cough. This cough comes on in attacks, during which the patient holds his breath and then takes a long inspiration with the characteristic stridulous sound.

In the mild cases the bronchitis is not severe, the paroxysms of cough not very frequent, and the patients suffer but little.

In other cases the paroxysms are so frequent and accompanied with so much vomiting, that the patient's health may suffer to a dangerous degree.

The worst cases are those which are complicated with broncho-pneumonia.

The disease lasts for from six to twelve weeks.

*Treatment.*—Inhalations of creosote, of carbolic acid, may be of service.

The bronchitis may be alleviated by the use of opium, ipecac, belladonna, or hydrocyanic acid; or by counter-irritants applied to the wall of the chest.

Applications of cocaine to the nose may be of service.

Strychnia, conium, arsenic, quinine, the bromides, valerian, asafœtida, and chloral are given as empirical remedies.

Change of climate is often of great service.

## MUMPS.

### (INFECTIOUS PAROTITIS.)

An infectious disease characterized by constitutional symptoms and inflammation of the salivary glands.

*Lesions.*—Either one or both parotid or submaxillary glands are inflamed. The inflammation only goes on to the point of congestion and swelling of the glands. Occasionally the testicles or the mammary glands are inflamed in the same way.

The poison of the disease has not been demonstrated. The disease is contagious from person to person.

*The period of incubation* is from fourteen to twenty-one days.

The symptoms may begin with the constitutional disturbances, or with the inflammation of the salivary glands, or with both together.

The constitutional symptoms are: fever, headache, nausea, restlessness, irritability, and prostration.

The local symptoms consist in the pain and swelling of the inflamed glands. With this there is often some pharyngitis.

The inflammation of the glands reaches its height in from three to six days, remains stationary for one or two days, and then subsides.

If only one parotid is inflamed, or if in both parotids the inflammation begins at the same time, the disease lasts about a week. If the parotids are inflamed successively, the disease is protracted for two weeks.

The complicating inflammations of the testicles or mammæ add very much to the discomfort of the patient and protract the disease.

Most cases of mumps run a mild course. There may, however, be feeble heart-action, or excessive fever, or delirium.

*Treatment* is directed to the alleviation of symptoms.

## ANÆMIA.

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In health, while the different constituents of the blood—the red cells, the white cells, and the plasma—are constantly undergoing change, destruction, and reproduction, their relative quantities continue the same. In exactly what way, and exactly where, the death and new formation of the blood-cells take place we do not definitely know.

In disease the blood may be changed by a diminution in the number of red cells, a diminution in the quantity of hæmoglobin, or an increase in the number of white cells.

When the change consists in a diminution in the number of red cells and in the quantity of hæmoglobin, the patient is said to suffer from "anæmia." If such changes in the blood are well marked, we may find in addition a variation in the form and size of the red cells, yellow spherical microcytes, and nucleated red cells.

It is evident that a diminution in the number of red cells must be due to a diminished new formation, or an increased destruction; but to tell which of these is the efficient cause in each case of anæmia is very difficult.

While our knowledge of anæmia is still so imperfect that we cannot tell certainly the relationship of its different forms, it is necessary, for purposes of prognosis and treatment, to adopt some form of classification. For this purpose I find it convenient to class all the cases of anæmia into four groups: 1, Secondary Anæmia; 2, the Primary Anæmia of Young Women; 3, Pernicious Anæmia; 4, Anæmia apparently intermediate in character between simple and pernicious anæmia.

### I. SECONDARY ANÆMIA.

Secondary anæmia is of very frequent occurrence. It is caused by Bright's disease, phthisis, malaria, cancer, uterine disease, any chronic and exhaustive disease, and hemorrhage. It

also occurs, especially in women, in persons whose general health is bad, although they have no definite disease.

In these patients the pallor of the skin and mucous membranes is usually very marked. The diminution in the quantity of hæmoglobin and the number of red cells is never very great. Of the other symptoms which may be present, it is always difficult to tell how many are due to the primary disease and how many to the anæmia.

In the treatment of these patients the essential parts are the treatment of the primary disease and the improvement of the patient's general health by food, climate, and mode of life. Iron and oxygen are only accessory parts of the treatment, and often of little or no service.

## 2. THE PRIMARY ANÆMIA OF YOUNG WOMEN.

*Synonym.*—Chlorosis.

*Causes.*—The disease occurs regularly in women between the ages of fourteen and thirty years. It is said by some authors that it never occurs in the male, but this certainly is an error. Young males are also affected by the disease, although in small proportion compared to the number of women affected. In women the disease is a very common one, and is observed both in persons living in cities and in those living in the country, among the rich and among the poor, in the midst of perfect health and after depressing conditions. It is one of the curious features of the disease that a strong, healthy girl, eating well, sleeping well, taking plenty of out-door exercise, can, without discoverable cause, become anæmic. We are ignorant of the causes of the disease and of the causes of the changes in the blood. Whether there is an increased destruction of red blood-cells and of hæmoglobin or a diminished production is uncertain.

*Symptoms.*—The most important symptom is the change in the composition of the blood. There is always a diminution in the quantity of hæmoglobin and usually a decrease in the number of red blood-cells. The red blood-cells have to be counted with the Thoma-Zeirs hæmacytometer, or with one of the other instruments made for that purpose, and we reckon 5,000,000 red blood-cells to the cubic millimetre as the standard in health. The estimation of hæmoglobin is easily made with the Fleischl hæmometer. The counting of the red blood-cells is tedious, and

the liability to error considerable. The estimation of the hæmoglobin is quickly and easily done, and answers alone for the larger number of cases. In many patients a doubtful diagnosis is settled at once by the estimation of the hæmoglobin. The loss of hæmoglobin is always marked, it falls to from twelve to seventy-five per cent. It is often, but by no means always, out of proportion to the loss of red blood-cells, thirty-eight per cent of hæmoglobin, for example, with 4,308,000 red cells. The ordinary rule, therefore, in these patients is to find the red blood-cells not much below 4,000,000 to the cubic millimetre, while the hæmoglobin is below fifty per cent. But it is by no means a rare exception to have the number of red cells below 2,000,000. A patient with 1,500,000 red blood-cells and twenty per cent. of hæmoglobin can be considered an advanced case of simple anæmia. As the patients improve under treatment, the number of red blood-cells and the quantity of hæmoglobin increase, the number of red blood-cells reaches the normal standard with considerable certainty ; but it is much harder to get the hæmoglobin above ninety per cent.

The skin and mucous membranes are usually distinctly pale, although seldom of the absolute whiteness that belongs to pernicious anæmia ; but it must be remembered that persons may look pale with a normal composition of the blood, and may look of good color although they are really anæmic. It is for this reason that the actual estimation of the hæmoglobin is of so much consequence.

The patients remain well nourished or they become somewhat emaciated. The loss of muscular strength and the indisposition to bodily or mental exertion are well marked. Irritability of temper, a loss of interest in ordinary occupations, neuralgic pains in different parts of the body, and a great variety of hysterical symptoms are often present. When a young girl complains that she cannot ride, or take long walks, or play tennis as she has been accustomed to ; when she is dull about her lessons, cross and peevish with her family, listless and headachy, we must always think of anæmia as a probable cause.

The heart's action is rapid, with dyspnœa on exertion. The dyspnœa increases as the anæmia progresses. In the extreme cases the patients cannot even get out of bed without fainting. There is often a systolic murmur diffused over the præcordial area, or with its maximum intensity at the second left intercostal

space, or at the apex. Occasionally the heart is enlarged, the enlargement disappearing as the patients recover from the anæmia. These cardiac symptoms are the direct result of the anæmia. We must not allow them to mislead us into the belief that the patients have either a disease or a neurosis of the heart.

The patients may be annoyed by a cough due to the condition of the pharynx or to hysteria. They may from time to time cough up small quantities of blood. It is not always easy to distinguish these patients from cases of commencing pulmonary tuberculosis.

Disturbances of the functions of the stomach are very frequently present and are often so marked as to attract more attention than does the anæmia by which they are caused. The patients complain of gastric pain, made worse by the ingestion of food, loss of appetite, nausea, vomiting, and vomiting of blood. It is of great importance not to mistake these patients for cases of catarrhal gastritis or of ulcer of the stomach, for it will be found that all the gastric symptoms disappear if the anæmia is relieved. The bowels are often constipated, perhaps not more so than is the case in young women who are not anæmic.

The urine is often turbid and alkaline. It may contain small quantities of albumin without disease of the kidneys. An anæmic patient with a little albumin in the urine and œdema of the legs or face may readily be mistaken for a case of nephritis.

The menstrual flow is usually light-colored, scanty, irregular, or stops altogether ; but it is sometimes in excess.

In the more severe cases there is an irregular febrile movement, the temperature higher in the afternoon, but not often higher than  $101^{\circ}$  F.

The cases vary as to the rapidity with which the symptoms are developed, their severity, their number, and the predominance of one or more symptoms over others. In the mild cases the patients hardly feel sick, and do not understand why bodily and mental exertion are becoming difficult to them. In the more severe cases the shortness of breath and muscular feebleness render the patients unable to work, while the nervous and gastric symptoms give them much annoyance. The worst patients cannot be distinguished from cases of pernicious anæmia. They are in bed, extremely feeble, of a ghastly white color, with fever, either delirious or stupid, bleeding from the mouth and from

the stomach, vomiting from time to time ; but even from this unpromising condition they can recover altogether.

In some patients the anæmia with its symptoms are developed very rapidly, sometimes even within a few days. More frequently they are developed slowly, so that several months elapse before the patient is much inconvenienced.

Occasionally we see women who have gone for three or four years before they have had to give up work, slowly getting more anæmic, but not adopting any plan of treatment.

*The prognosis* of the disease depends upon its treatment. If properly treated the patients recover, but the treatment must be continued for a long time and relapses must be expected. The only cases which are likely to prove fatal are those with vomiting of blood, and those in which the hæmoglobin is less than twenty per cent. and the number of red blood-cells less than two million to the cubic millimetre.

*Treatment.*—In the mild cases without much prostration or gastric disturbance, all that is necessary is the ingestion of iron. The best preparation of iron for all cases of anæmia is the sulphate, the best time to take it is after meals, and the quantity should be from twelve to twenty-four grains in the twenty-four hours.

In the cases with pain in the stomach, nausea, and vomiting, the food must be restricted, but the food and iron can be taken by the stomach even if the patients vomit blood. It is, however, necessary to keep the patients in bed.

In the advanced cases with marked dyspnœa and feebleness, it is important at first to keep the patients in bed, with massage in addition to the use of iron.

There are some persons who at first either cannot or will not take iron. In these persons there is an advantage in the use of inhalations of oxygen gas continued for ten minutes two or three times a day. As soon as they have learned to take iron enough the oxygen becomes unnecessary.

In all the cases of anæmia it is absolutely necessary for the bowels to move every day. If they do not move the iron treatment will not exert its specific effects in changing the composition of the blood.

It is necessary to examine the blood of these patients every week, so as to be sure that the quantity of hæmoglobin and the numbers of the red blood-cells are really increasing as they ought to do.



## 3. PERNICIOUS ANÆMIA.

*Synonyms.*—Idiopathic Anæmia ; Essential Anæmia.

*Lesions.*—In some of the persons who have pernicious anæmia there are found after death : cancer of the stomach, chronic gastritis, degeneration or atrophy of the gastric tubules, the anchylostoma duodenale or bothriocephalus latus in the intestine, or chronic nephritis. It is believed by some that such lesions may cause pernicious anæmia ; by others that their association in the same person is accidental.

As the results of the anæmia we may also find fatty degeneration of the wall of the heart, degeneration of the hepatic cells, the renal cells, the walls of the arteries and capillaries.

In the blood the number of red cells is very much diminished. There may be less than one million red cells to the cubic millimetre. Many red cells are found of abnormal size or shape, or containing nuclei. Yellow, spherical microcytes, about one-fourth the size of the red blood-cells, are often present. The entire quantity of hæmoglobin is diminished often below twenty per cent., but not the quantity belonging to the red cells which are left in the blood. The individual red cells are still rich in hæmoglobin.

In the marrow of the long bones there are found nucleated red blood cells and cells enclosing old red cells.

Hunter has lately called attention to the condition of the liver. He has found in it, with pernicious anæmia, always an excess of iron. The iron is in the form of pigment granules in the liver-cells, which form the outer two-thirds of each lobule.

*Nature of the Disease.*—Two entirely different views are held concerning the nature of pernicious anæmia. One view is that it is simply an advanced form of anæmia, which can be produced by a variety of causes. The other that it is a primary disease, entirely distinct from other forms of anæmia.

The study of the disease made by Hunter has led him to the following conclusions :

1. Pernicious anæmia is a special disease.
2. Its essential pathological feature is an excessive destruction of blood-cells.
3. The most constant anatomical change is the presence of an excess of iron in the liver.

4. This condition of the liver distinguishes pernicious anæmia from symptomatic anæmia.

5. The disintegration of the red cells is effected chiefly in the portal circulation, especially in the spleen and liver. The destruction is effected by poisonous agents absorbed from the intestinal tract.

*Symptoms.*—The disease regularly begins slowly and insidiously, much less frequently rapidly.

The color of the skin and mucous membranes becomes pale, often with a dusky or yellow tinge.

There is gradual loss of muscular and mental strength.

The functions of the stomach and intestines are disturbed.

There is palpitation of the heart, dyspnœa on exertion, attacks of syncope, a systolic murmur heard at the apex or in the second left interspace.

There may be hemorrhages into the retina, into the skin, from the nose, mouth, bronchi, stomach, or intestines.

At some time in the course of the disease there is an irregular febrile movement.

After a time the patients lose flesh as well as strength, and the entire condition is that of very great feebleness.

The disease lasts for weeks, months, years. The patients steadily get worse, but often with intervals of considerable improvement. The cases which run their course within a few weeks are the least frequent.

The prognosis is bad.

*Treatment.*—The most efficient drug in the treatment of the disease is arsenic, which must be given in considerable doses. Iron, oxygen, feeding, and nursing are accessory measures.

#### 4. INTERMEDIATE ANÆMIA.

Anæmia which clinically holds an intermediate place between simple anæmia and pernicious anæmia.

I describe under this indefinite name a group of cases of which I see a considerable number, and which I am, therefore, for my own convenience, obliged to arrange into some sort of a class. I admit that many of these cases may possibly be examples of a mild form of pernicious anæmia, and that it is not easy to distinguish some of them from the secondary anæmias.

The patients are both males and females between the ages of

forty and sixty years. In some of them there is a history of overwork ; in some of them it takes some time to show that they have no obscure malignant disease ; but in many the disease comes on without any discoverable cause.

The blood shows a diminution in the quantity of hæmoglobin and the number of red blood-cells, but not to an extreme degree. The hæmoglobin is not usually below fifty per cent., nor the red blood-cells below 3,000,000 to the cubic millimetre. The relative diminution of the hæmoglobin and of the red cells is more like that of simple anæmia than that of pernicious anæmia.

The change in the color of the skin, of the mucous membranes, and of the sclerotic is very marked. These are of a whiteness greater than one would expect with the degree of anæmia. In this way the cases resemble those of secondary anæmia. Some of the patients lose flesh, others do not. Dyspnœa is not a marked feature. The functions of the stomach and intestines are often disturbed. The most marked feature is the loss of muscular strength and the indisposition to mental exertion. The patients improve greatly under treatment, but yet never get quite well. They continue to live for many years. Most of them have passed out of my observation while still in good condition ; some have died from intercurrent diseases ; a single one died as if with pernicious anæmia.

*Treatment.*—These patients are improved by change of climate, exercise, diet, and by any measures which improve their general health. Small doses of arsenic, combined with large doses of iron, increase the quantity of hæmoglobin and the number of red blood-cells.

If we compare the different forms of anæmia as regards their treatment, we find that :

1. In secondary anæmia attention to the general health is of principal importance, while iron and arsenic are of much less consequence.
2. In the primary anæmia of young persons iron in large doses is a specific.
3. In pernicious anæmia arsenic in large doses gives the best results.
4. In the intermediate form of anæmia we do best with arsenic in small doses, iron in large doses, and attention to the general health.

## LEUKÆMIA.

This is a disease characterized by diminution in the number of red blood-cells and the quantity of hæmoglobin, increase in the number of white blood-cells, enlargement of the spleen, lymphatic glands, and marrow of the bones.

*Lesions.*—The specific gravity of the blood is lowered. Its color is light-red or even whitish, looking like pus and blood mixed. The number of the red cells is diminished. The white blood-cells are very much increased in number ; the increase in the number of the larger white cells is especially marked in the cases in which the spleen is enlarged.

The marrow of the bones is hypertrophied, of yellow or red color, with an increased growth of both the cells and stroma.

The spleen is in most cases much enlarged, this enlargement being a simple hypertrophy.

The lymphatic glands are often hypertrophied, or tumors composed of lymphatic glandular tissue are formed in the liver, kidneys, lungs, stomach, intestines, or peritoneum.

There may be extravasations of blood in any part of the body. There may be an inflammation of the retina resembling that which complicates Bright's disease.

There may be fatty degeneration of the walls of the heart.

*Causes.*—The disease has been observed in persons between the ages of eight weeks and seventy years. It occurs most frequently between the ages of thirty and forty years. It is more frequent in man than in woman.

In some patients the disease seems to be secondary to malarial poisoning, to syphilis, to blows on the spleen, or to starvation.

*Symptoms.*—The cases with hypertrophy of the spleen, the lymphatic glands, and the marrow are the most common. Next in frequency are those with hypertrophy of the spleen and marrow ; next, those with hypertrophy of the lymphatic glands and marrow ; while the least frequent are those with hypertrophy of the marrow alone.

The symptoms due to the changes in the blood are present in all the cases.

The symptoms begin and go on slowly and gradually ; either the change in the patient's general condition or the enlargement of the spleen or glands may first attract attention.

The change in the color of the skin and mucous membranes is usually well marked, the patient becoming extremely white. Occasionally, however, the color remains natural for a long time.

There is a gradual loss first of strength, and then of flesh. The mental faculties may remain for a long time unimpaired, or they may become dull and sluggish, with peevishness and irritability of temper.

The eyesight may be impaired by the complicating retinitis.

The heart's action becomes feeble, irritable, with a systolic murmur, dyspnœa, and liability to attacks of syncope.

Priapism is a curious symptom which has been present in a large number of cases.

There may be pain after eating, nausea, and vomiting.

Menstruation is scanty and irregular. There may be a moderate amount of subcutaneous dropsy.

The temperature remains normal, or from time to time there is an irregular febrile movement.

Hemorrhages are common—from the nose, gums, bronchi, stomach, intestines, uterus, bladder. The largest bleedings are those behind the peritoneum, which may cause death within a short time.

The enlargement of the spleen, glands, and liver is easily recognized.

The disease regularly lasts for months or years, with periods of exacerbation and of improvement.

*The prognosis* is bad, but a few cases of cure have been reported.

*Treatment.*—The drugs most frequently employed are arsenic, phosphorus, and iron. The hypertrophy of the spleen has been treated by electricity, interstitial injections, and extirpation.

#### PSEUDO-LEUKÆMIA.

*Synonyms.*—Anæmia Lymphatica; Hodgkin's Disease.

A disease characterized by a progressive decrease in the number of red blood-cells, an enlargement of the lymphatic glands, and new growths of lymphatic glandular tissue.

*Lesions.*—The changes in the blood consist simply in a diminution in the number of red blood-cells, without any increase in the number of white cells.

Of the lymphatic glands, one or more groups are enlarged in

the following order of frequency: the cervical glands, the axillary, the inguinal, the retro-peritoneal, the bronchial, the mediastinal, and the mesenteric. Of these groups of glands those on one or on both sides of the body may be involved. The enlargement of the glands is a simple hypertrophy, with an excess either of the stroma or of the cells.

The new growths of lymphatic glandular tissue are found in the spleen, the liver, the œsophagus, the stomach, the intestines, the peritoneum, the kidneys, the lungs, the pleura, the ovaries, the testicles, the dura mater, and the skin.

The marrow of the bones is changed in the same way as in leukæmia.

*Causes.*—The disease is more frequent in males than in females, in the proportion of three to one. It has been observed in persons between the ages of one and seventy years, but it is most frequent in early and late adult life.

*Symptoms.*—Both the changes in the glands, the new growths of glandular tissue, and the changes in the blood contribute to the symptoms.

The enlarged cervical glands may press on the pharynx, the larynx, the carotids, and the veins of the neck.

The enlarged intra-thoracic glands may press on the trachea, the œsophagus, the bronchi, and the descending vena cava.

The enlarged glands in the abdomen may cause pain, they may press on the common bile-duct or the portal vein.

The enlarged glands in the pelvic and iliac regions may cause pain in the thigh or along the track of the sciatic nerve, and œdema of the leg.

The new growths in the liver and spleen are accompanied with the enlargement of these organs.

The changes in the blood produce symptoms like those of pernicious anæmia:

Feeble heart-action with dyspnœa on exertion; bodily and mental weakness; dropsy; hemorrhages; nervous and hysterical symptoms; an irregular febrile movement; pain and tenderness over the bones. Some of the patients, toward the close of the disease, develop delirium, general convulsions, or coma.

The disease begins either with the symptoms belonging to the anæmia or with those belonging to the glands. The patients slowly get worse, both sets of symptoms becoming more and more marked, but yet one usually predominating.

The patients finally die worn out, feeble, and emaciated ; or from the pressure of the enlarged glands on the blood-vessels or air-passages ; or with cerebral symptoms ; or from some inter-current disease.

*Treatment.*—The most efficient methods of treatment seem to be the removal of the enlarged glands by operation and the internal administration of arsenic or of iodine in considerable doses.

#### ADDISON'S DISEASE.

*Definition.*—A disease characterized by anæmia, general languor and debility, feeble heart-action, irritability of the stomach, a peculiar change in the color of the skin, and disease of the supra-renal capsules (Addison).

Later authors have included with the disease cases of diseased supra-renal capsules and anæmia, without pigmentation of the skin ; and cases of pigmented skin and anæmia, without disease of the supra-renal capsules.

*Lesions.*—The regular change in the supra-renal capsules is a tubercular inflammation, which results in the conversion of the capsules into masses of fibrous tissue and cheesy matter.

Less frequently the capsules have been found atrophied, or absent, or the seat of malignant disease.

The change in the skin consists in a deposition of pigment in the deeper epithelial cells and in the connective-tissue cells of the cutis.

The spleen is sometimes enlarged.

In the abdominal sympathetic there have been found an increase of fibrous tissue and a degeneration of the nerve-fibres and cells.

In the blood there is sometimes a diminution of the number of red cells and the quantity of hæmoglobin.

*Nature of the Disease.*—While we are still very ignorant of the nature of the disease, there is a general impression that the disease of the supra-renal capsules is to be looked on as a causative lesion.

G. Tizzoni (1889) has published the results obtained from a long series of experiments on animals. He concludes :

1. In rabbits the destruction of one or both supra-renal capsules causes death.

2. Death takes place after a few hours or many months.

3. Before death, especially in the longer-lived rabbits, symptoms are developed like those of Addison's disease—pigmentation of the skin, emaciation, loss of strength, stupor, convulsions.

4. After death lesions are found in the cerebrum, cerebellum, cord, and peripheral nerves. In the cerebrum and cerebellum the changes are diffuse. In the cord they are most marked in the lower cervical and upper dorsal regions. They begin in the central canal and extend through the gray and white commissures to the anterior and posterior horns.

The lesion is a degeneration of the nerve-fibres and ganglion-cells, with congestion, hemorrhages, and infiltration, with leucocytes.

*Causes.*—The disease has been observed in persons between the ages of eleven and fifty-eight years. It is more common in males than in females. We know nothing concerning its causation.

*Symptoms.*—There is a gradual loss of muscular strength. The mind becomes dull, apathetic, listless; the temper is often irritable and peevish. The heart's action is feeble and rapid, with dyspnœa on exertion and liability to attacks of dyspnœa. The functions of the stomach are often disturbed, with pain, nausea, and vomiting. There may be irregular pains in the loins, epigastric, and hypochondriac regions.

As the disease goes on, the mind becomes more feeble, and the patients less able to take care of themselves. They may suffer from vertigo, from feelings of numbness in different parts of the body, or portions of the skin may be anæsthetic.

As the anæmia is developed, the skin and mucous membranes become pale.

The characteristic brownish discoloration appears in those parts of the skin which are usually uncovered by clothing, and in the regions where the skin is naturally darker. The mucous membrane of the lips, gums, and tongue may be discolored in the same way.

In the last stages of the disease the patients are exceedingly feeble; stupor, delirium, general convulsions, or coma may be developed.

The patients die from asthenia, in an attack of syncope, or in convulsions.

The disease is protracted for months or years, with periods



of remission and of exacerbation, but the patients always finally become worse.

Rarely, the constitutional symptoms are very slight until a few days before death.

The discoloration of the skin may exist for months or years before any of the other symptoms, or the general symptoms may last for a long time before any discoloration of the skin is established.

The *treatment* of Addison's disease is directed entirely to the alleviation of symptoms and the comfort of the patient. For the disease itself no treatment is known.

## HYDROPHOBIA.

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An infectious disease belonging to dogs, foxes, and wolves, and sometimes communicated from them to man and to other animals.

The poison of the disease exists in the saliva and the spinal cord. Its micro-organism has not yet been demonstrated. In man the disease is only acquired by inoculation from the bite of a rabid animal. In animals it appears to originate without discoverable cause. The disease may be given to animals experimentally by inoculation with the saliva or the spinal cord.

About half of the human beings who are bitten by rabid animals escape the disease.

*Lesions.*—The lesions are in the cerebro-spinal system, the blood-vessels are congested, there is perivascular exudation of leucocytes, and there are minute hemorrhages. According to Gowers, these are particularly intense in the medulla. The pharynx, larynx, trachea, bronchi, and stomach are congested.

*The period of incubation* lasts for from only a few days up to three months. Horsley states that the length of time depends upon the following factors :

- a.* Age. The incubation is shorter in children than in adults.
- b.* The part infected. The rapidity of onset of the symptoms is greatly determined by the part of the body which may happen to have been bitten. Wounds about the face and head are especially dangerous ; next in order in degrees of mortality come bites on the hands, then injuries on the other parts of the body. This relative order is, no doubt, greatly dependent upon the fact that the face, head, and hands are usually naked, while the other parts are clothed.
- c.* The extent and severity of the wound. Punctured wounds are the most dangerous ; the lacerations are fatal in proportion to the extent of the surface afforded for absorption of the virus.
- d.* The animal conveying the infection. In order of decreasing severity come : first, the wolf ; second, the cat ; third, the

dog ; and fourth, other animals. Only about fifteen per cent. of those bitten by rabid dogs become affected by the disease.

*Symptoms.*—1. The premonitory stage, which lasts for one or two days. The patients complain of headache, irritability, apprehension of evil, sleeplessness, increased sensibility to light and noise, loss of appetite, huskiness of the voice, commencing difficulty in swallowing, a slight rise of the pulse and temperature, and pain in the cicatrix of the bite.

2. The convulsive stage. First, the muscles of deglutition are easily excited to involuntary and painful contractions. Then the muscles of respiration, and the voluntary muscles throughout the body are affected in the same way. The saliva is increased in quantity, and the patients are constantly spitting it out. They vomit the little food they are able to swallow. There is venous congestion of the skin. There is violent delirium with hallucinations. The heart's action becomes rapid and feeble. The patients die exhausted in from one to ten days. The incessant convulsive movements and violent delirium give a terrible clinical picture. The temperature may rise from  $100^{\circ}$  to  $103^{\circ}$  F.

3. The paralytic stage. In rodents the preliminary and furious stages are absent as a rule, and the paralytic stage may be marked from the outset—dumb rabies. During this stage the patients become quiet, the spasms no longer occur, there is gradual unconsciousness, and the heart's action becomes more and more feeble.

*Treatment.*—The bite of a rabid animal should be at once excised or freely cauterized.

During the period of incubation inoculation with attenuated virus from the spinal cords of rabbits, after the method of Pasteur, has given good results.

During the convulsive period morphine and chloroform may be used very liberally.

#### FALSE HYDROPHOBIA.

This is a curious affection, which closely resembles hydrophobia, but is really nothing more than a neurotic or hysterical manifestation. A nervous person bitten by a dog, whether rabid or not, develops after several months symptoms resembling those of rabies. These attacks last longer than those of true rabies, and are amenable to treatment.

## TRICHINOSIS.

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This is an infectious disease belonging to swine. It is due to the introduction into the body of a species of worm—the trichina.

In man the disease is acquired by eating swine's flesh which contains living trichinæ.

When flesh containing trichinæ is taken into the stomach, the trichinæ are set free and pass into the small intestine. There they become mature within two or three days, and the females bring forth living young five days later.

The trichinæ are of the shape of thread-like worms, male and female. The male are  $1\frac{1}{2}$  mm. long, the female 3 to 4 mm. long. The young are born alive on the seventh day after the ingestion of the flesh containing trichinæ.

The young trichinæ soon leave the small intestine and find their way into the voluntary muscles. Here they increase somewhat in size and become enclosed in a connective-tissue capsule. Enclosed in this capsule the worm remains quiet but alive for years. Finally it dies, and the capsule becomes thickened and infiltrated with lime.

*The symptoms* vary with the number of trichinæ introduced into the stomach and born in the small intestine.

A small number of trichinæ produce very slight symptoms.

A large number of trichinæ produce severe symptoms.

From a few hours to several days after the ingestion of the infected meat there are disturbances of the stomach and bowels, pain, vomiting, and diarrhœa, with prostration.

About the tenth day begins the emigration of the young worms from the intestine and their lodgement in the muscles. While this is going on the muscles are swollen, contracted, tender, and painful; there is subcutaneous œdema in different parts of the body; there are irregular eruptions of herpetic, petechial, or pustular form; there is a continued fever with progressive emaciation; the patients pass into the typhoid state; there may be a complicating bronchitis or broncho-pneumonia.

The disease lasts from eleven to one hundred and twenty

days. It is often fatal. In the cases which recover convalescence is protracted.

*Treatment.*—The preventive treatment consists in an efficient inspection of the muscles of all swine that are slaughtered and in the thorough cooking of all swine flesh that is eaten.

During the first seven days after the ingestion of infected flesh it is possible to get rid of some of the trichinæ by vomiting and purging the patients.

## ANTHRAX.

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(MALIGNANT PUSTULE. MALIGNANT ŒDEMA. MILZBRAND.)

An infectious disease belonging to cattle, sheep, and horses, and which is communicated from these animals to man.

The characteristic micro-organism is a bacillus which is found in great numbers in the blood, and which can be cultivated and inoculated.

These bacilli may live for a long time in the grass and on the surface of the ground. They may originate on marshy soils, or may be brought there by diseased cattle. Cattle acquire the disease from food and air contaminated with the bacilli.

In man the disease is acquired by inoculation, by inhalation, or by the alimentary canal. Inoculation is effected by handling infected hides, hair, wool, flesh, or instruments. It is said that it can also be done by flies and mosquitoes. The inhalation is of the dust from infected hides, hair, or wool. The introduction into the stomach of infected flesh can give the disease.

*Symptoms.*—The disease in man regularly follows one of three forms:

1. *Malignant Pustule.* This is the most common form. It is acquired by inoculation. There is a period of incubation lasting from a few hours to fourteen days.

At the point of inoculation there is first a little swelling looking like a flea-bite, with a central black point. This swelling becomes larger, a vesicle is formed on it, then there is a central dry necrosis surrounded by a thick, swollen, red zone looking like a carbuncle, and around this the skin is swollen and œdematous. The general symptoms are developed from forty-eight to sixty hours after the appearance of the pustule. There are fever, prostration, and the rapid développement of the typhoid state.

The disease lasts from two to thirty days. It is very apt to prove fatal.

2. *Malignant Oedema.* This is acquired by eating infected meat or breathing infected air.

After eating infected flesh the symptoms begin in from eight to forty-eight hours.

The patients have chills, nausea, vomiting, pain in the abdomen, and great prostration. After hours or days a subcutaneous oedema of some part of the body is developed.

This form of anthrax is very fatal.

3. *Intestinal Anthrax.* This is acquired by eating infected meat.

The patients suffer from prostration, pains in the head and limbs, pains in the abdomen, vomiting, bloody diarrhœa, rapid breathing, restlessness, stupor, convulsions, high or low temperature, bleeding from the mouth. In the more protracted cases carbuncles are formed on the arms or head. This form of the disease is very fatal; the patients die in from one to seven days.

*Treatment.*—Preventive treatment is directed to cattle and sheep. Diseased animals are destroyed, suspected animals are isolated, and inoculations with attenuated virus have been practised with success on a large scale.

In malignant pustule the site of inoculation should be destroyed by caustics or the actual cautery, and the bichloride of mercury may be sprinkled over the exposed surface. The subcutaneous connective tissue around the pustule may be injected with solutions of corrosive sublimate or of carbolic acid. The local application to the pustule of powdered ipecac mixed with water, and at the same time the internal use of ipecac in five-grain doses every three hours, is highly recommended.

#### GLANDERS—FARCY.

These names are given to an infectious disease of the horse which is sometimes communicated to man. The disease follows two forms as to the locality of its inflammatory lesions. There is an inflammation of the mucous membrane of the nose, with the formation of nodules—glanders; or an inflammation of the lymphatic glands, with the formation of nodules in the skin—farcy.

*Causes.*—The micro-organism of the disease was discovered by Löffler and Schütz. It is a short bacillus not unlike that of tubercle. Human beings become affected by contact with dis-

eased animals, usually by inoculation on an abraded surface of the skin. The contagion may also be received on one of the mucous membranes.

*Lesions.*—The essential lesion is the formation of the tumors composed of round cells in the mucous membranes and in the skin, sometimes also in the viscera. These nodules have a disposition to become necrotic and soften, and so form ulcers. Besides the formation of these nodules, there is inflammation of the mucous membrane of the nose and of the lymphatic glands.

*Acute Glanders.*—*Symptoms.* The period of incubation is rarely more than three or four days. At the point of infection there are redness, swelling, and lymphangitis. Within a few days the mucous membrane of the nose becomes inflamed and coated with a muco-purulent discharge, while nodules are formed in its stroma. The nodules soon soften and form ulcers. An eruption of papulæ, which rapidly become pustules, breaks out over the face and about the joints. This eruption has been mistaken for that of variola. The nose becomes much swollen, the discharge from it is abundant and offensive. The lymphatic glands in the neck are swollen. Inflammation of the lungs is often developed. There is a febrile movement throughout. The disease runs its course within ten days and is invariably fatal.

*Chronic Glanders* is of rare occurrence, and may be mistaken for a chronic catarrhal inflammation of the nose. There is a discharge of mucus from the nose and the formation of chronic ulcers in its mucous membrane. The disease lasts for a number of months, and recovery is possible.

*Acute Farcy.*—At the point of inoculation a little tumor is formed, surrounded by inflamed skin. This soon degenerates and forms an unhealthy ulcer. Then the adjacent lymphatic vessels and glands become inflamed, nodules are formed in the skin, the joints become inflamed, and abscesses are formed in different parts of the body. The patients give the symptoms of virulent poisoning, they have headache, delirium, stupor, a febrile movement, rapid emaciation, and great prostration. The disease runs its course in from twelve to fifteen days, and is fatal in a large proportion of the cases.

*Chronic Farcy* is characterized by the presence of localized tumors, which are usually situated on the extremities. These



tumors break down and form abscesses and ulcers, but without inflammation of the lymphatics. The disease may last for months or years. Death may result from pyæmia or from acute glanders. Recovery is possible.

*Treatment.*—The original wound or tumor should be cut out or destroyed by caustics.

## ACTINOMYCOSIS.\*

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The disease belongs to the class of infective granulomata. It is a chronic inflammation excited by the presence of a special microphyte, with the production of new tissue composed of a stroma, round cells, epithelioid cells, and giant cells, which closely resemble tubercle tissue. The new tissue forms tumors of varying size, which often degenerate and suppurate.

The disease has existed for a long time in cattle under a variety of names; it is only later that it has been recognized in human beings. It has also been observed in pigs and horses.

It is most frequently seen in young cattle between the ages of one and three years, but it may occur at any age.

It is probable that the micro-organism gains access to the system through wounds of the skin and mucous membranes, or through carious teeth. The cow-sheds, pastures, and drinking-tanks may be infected by the discharges from diseased animals.

Actinomycosis of the lips occurs in the form of rounded nodules, or flat, wart-like growths, or ulcers, or polypoid growths of considerable size.

In the upper and lower jaws the disease seems to originate in carious teeth and then extends to the bone, which becomes the seat of chronic inflammation with enlargement.

The tongue is enlarged, is the seat of a diffuse inflammation, and is studded with nodules which may suppurate or ulcerate.

In the pharynx the growth assumes the form of submucous, polypoid tumors. Similar growths are found in the larynx and trachea.

In any part of the body tumors may be found in the deeper layers of the skin, which may suppurate.

In the lungs are found nodules which look like miliary tubercles.

The actinomyces may be seen with the naked eye in the mucopurulent discharge or in the scrapings from a growth. The tufts

\* Report of the Agricultural Department. London, 1889. Crookshank.

of the fungus vary in size from that of a grain of sand to that of a pin's head. They are made up of aggregations of club-shaped rods which form a sort of rosette. They may be calcified.

In the human subject actinomycosis affects the tissues of the mouth and neck and the jaws, producing lesions which come under the care of the surgeon ; and the lungs and the gastro-intestinal tract, which come under the care of the physician.

Thirty-three cases of actinomycosis of the lungs have been collected by Dr. Hodenpyle, from which the following account has been prepared :

The ages of the patients varied from nine to sixty-three years ; the majority of the patients were young adults.

The symptoms were those of a subacute broncho-pneumonia : cough and muco-purulent sputa, sometimes fetid, sometimes containing actinomyces ; the physical signs of bronchitis and of consolidation of part of one lung ; an irregular febrile movement, and progressive loss of flesh and strength. In some of the patients the formation of abscesses in the wall of the chest, so that they were mistaken for cases of empyema.

The average duration of the disease was ten months.

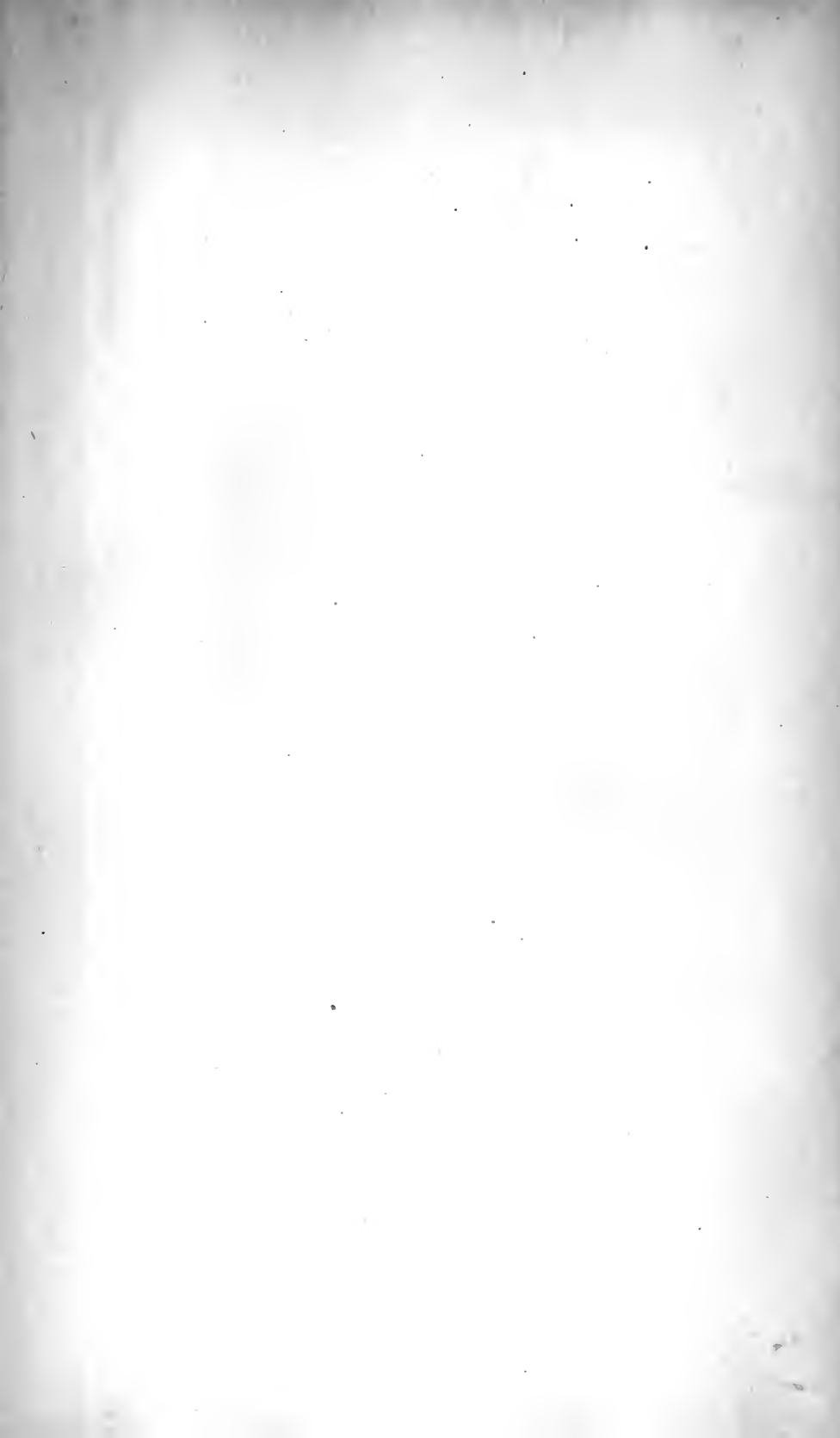
*Lesions.*—Usually only one lung is involved. The lesions are those of a broncho-pneumonia of peculiar form. The bronchi contain pus and actinomyces. Their walls are thickened by a growth of granulation-tissue or by little abscesses. Polypoid growths of granulation-tissue project inward from the walls of the bronchi, and irregular masses of organized connective tissue are found in their cavities. The walls of the air-spaces are thickened, their cavities are filled with epithelium or organized connective tissue. In some places are masses of granulation or connective tissue obliterating the air-spaces.

*Diagnosis.*—In all cases of broncho-pneumonia of irregular type the sputa should be examined for actinomyces.

*Prognosis.*—Nearly all the cases thus far reported have proved fatal.

*Treatment.*—The indications seem to be to use systematic inhalations of creosote, and to open and scrape out the abscesses in the wall of the chest.





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